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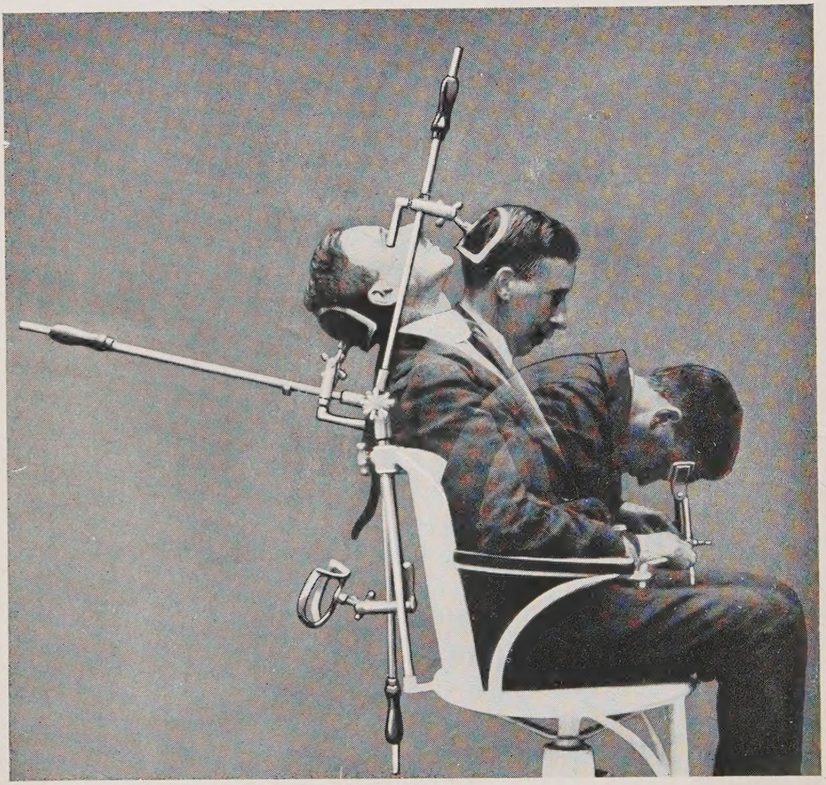

HEAD 60° BACK; HEAD 30° FORWARD; HEAD 120° FORWARD

# EQUILIBRIUM AND VERTIGO

By

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*WITH AN ANALYSIS OF PATHOLOGIC CASES*

By

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ADOPTED AS STANDARD FOR
MEDICAL DIVISION, SIGNAL CORPS, AVIATION SECTION
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DR. CHARLES K. MILLS,

DR. B. ALEXANDER RANDALL,

DR. T. H. WEISENBURG.

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# PREFACE

The studies presented in this book were originally undertaken at the suggestion of Dr. T. H. Weisenburg. Most of the work was done at the University of Pennsylvania under the guidance of Dr. B. Alexander Randall, whose encouragement will always be affectionately remembered. A sub-department of "Neuro-Otology" was created, in which Dr. Lewis Fisher, Dr. Seth A. Brumm and the writer conducted experiments which constituted the foundation for this study. Acknowledgment is made of the sympathetic co-operation and assistance of Dr. Charles K. Mills, who, with Dr. Weisenburg, guided us from the Neurologic standpoint; and also to Dr. William G. Spiller for his unstinted interest and advice. The Neurologic Chapters, XII and XIII, constitute the presentation of the direct teachings of Drs. Mills and Weisenburg.

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The studies of the relation of the ear to the eye were undertaken with the help of Dr. H. Maxwell Langdon, during a period of four years. The studies in syphilis were originally undertaken in co-operation with the late Dr. Alexander A. Uhle and later with his associate, Dr. William H. Mackinney.

The dissections of the brain were made by the eminent anatomist, Dr. Addinell Hewson, and the stereoscopic photographs,* although taken by the author, were made possible by the equipment and expert guidance of Dr. Matthew H. Cryer.

The moving pictures were taken and arranged through the courtesy of Mr. S. Lubin, who has shown a most sincere interest in the advancement of the use of moving pictures as a means of medical teaching and research.

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* A duplicate set will be found in the back of the book for use with the stereoscope.

The diagrams and photographs were perfected by the painstaking work of W. H. Hoedt.

Thanks are due to Emilie Austin Jones for original suggestions regarding the outline of the book and the arrangement of the chapters, and to Lillie B. Lynn for years of daily effort in systematizing the numberless details incidental to the production of this book.

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Isaac H. Jones

Office of Chief Surgeon,
Aviation Section, S. C., U. S. Army,
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April, 1918

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# PART I

## THE PRACTICAL USES OF A STUDY OF THE INTERNAL EAR

# EQUILIBRIUM AND VERTIGO

## CHAPTER I

## NEURO-OTOLOGY

Any work in physiology may be looked on as an interesting study, but from the standpoint of most of us in the practice of medicine, any research work attains a much more real importance when it can be directly applied to clinical work and proves to have an immediate, practical usefulness. When we consider the intimate relation of the ear to the rest of the body through the nervous system, it at once becomes evident that a wealth of information may be obtained from such a study. Neuro-otology is a suitable name for this study, in that it consists of an analysis of the internal ear and its associated intracranial nerve pathways. Although it is obviously an otologic study, and is useful in the analysis of ear conditions, yet it has a much broader significance in its usefulness as a means of providing data in general medical and surgical diagnosis and treatment. It not only opens up a new field for investigation in physiology, therefore, but furnishes information of diagnostic value in the every day examination of patients.

The value of the information gathered from the study of the eye and nerve-pathways from the eye is universally conceded, but it is only in the past few years that similar possibilities in ear-study have come to light. The eye specialist not only relieves ills strictly confined to the ocular tissues themselves conjunctivitis, refractive errors, muscle imbalance and cataract but he is asked by men in other fields of medical and surgical work for valuable information concerning intracranial conditions, renal and vascular disease, metabolic disturbances, varied evidences of intoxications, and is also asked to interpret their significance in regard to the general health of the patient. As a result of this sort of service, ophthalmology has for years taken its proper place in general medicine. Otology is now entering into a similar field of usefulness. The value of ear-study, however, is not yet generally recognized. The internist does not turn to the otologist for the analysis of the cause of vertigo. The syphilologist does not yet recognize the uses of ear-tests in detecting early involvement of the central nervous system. The neurologist does not yet catalog among his indispensables the accurate data otology can furnish concerning the degree of function of the VIII Nerve, in a differentiation between labyrinth and intracranial lesions and in intracranial localization. The general surgeon does not recognize that a lesion within the internal ear may simulate intra-abdominal carcinoma, nor does the cranial surgeon yet recognize the value of ear-examination in helping him to diagnosticate and locate intracranial lesions.

This newer usefulness of otology is made possible by a study of the vestibular or "balance" portion of the ear. The internal ear has long been thought of merely as the organ of hearing; viewed in this light its affections were mainly of local significance. Because of its supposedly limited connections with the nervous system, its phenomena were seldom regarded as the expression of any general disorder. Now, however, it is recognized that the internal ear consists of two organs instead of one—the cochlea, which is the organ of hearing, and the kinetic-static labyrinth, which is the organ of equilibration. It is this kinetic-static portion of the ear, consisting of the utricle, saccule and the three semicircular canals, that has been through all the years a "terra incognita." It is only in the past few years that the function of the vestibular portion of the labyrinth has been carefully studied, pre-eminently by the Vienna group of otologists, to whom we are indebted for the new methods of testing the internal ear. Robert Barany received the Nobel prize in 1915 for this work, and he is to be regarded as the pioneer in the clinical application of the study of the relations of the ear and the central nervous system. The internal ear as the chief organ of balance or equilibration at once assumes an importance far greater than the ear as an organ of hearing. As an equilibratory organ it must be richly supplied with nerve-pathways connecting it intimately with many nerve-centres which in their ultimate distribution affect the entire body. It is this larger mechanism that has opened up new possibilities for the clinician. The new ear-tests stimulate not only the ear itself but this entire widely distributed nerve apparatus. A stimulus applied to the ear produces phenomena very remote from the ear itself; parts of the body even as remote as the foot may be affected by such stimulation. There is in fact no portion of the body-musculature that is unaffected by stimulation of the vestibular labyrinth. It is evident that all these parts of the body can be affected only because of nerve-pathways connecting them with the ear. When stimulation of the ear produces the expected normal phenomena it demonstrates that these particular pathways are intact; conversely, an absence of normal responses indicates an impairment by disease of the ends or lines of these pathways.

The ear-tests consist of stimulating the semicircular canals; this is done by revolving a person in a turning-chair (Fig. 1), or by douching the ears with either cold or hot water (Fig. 2), or by applying the galvanic current to the ear. Such ear-stimulation produces certain definite phenomena—a rhythmic jerking of the eyes known as nystagmus, and a subjective sensation of turning which may be termed a systematized vertigo. The two distinct phenomena then are nystagmus and vertigo. Because of this vertigo, a patient falls in a definite direction, and also when, blindfolded, he attempts to find with his finger or his foot an object he has previously touched, he is unable to find it but "past-points" to the right or left, above or below, depending on the direction of the vertigo. These various phenomena are invariably present in normal people, and furthermore always follow definite laws. For example, turning the patient to the right with the head in the upright position, stimulating both "horizontal" semicircular canals, produces a horizontal nystagmus to the left, a subjective sensation of turning to the left, and a "past-pointing" to the right. Douching the right ear with cold water, head upright, stimulating the vertical canals, produces a rotary nystagmus to the

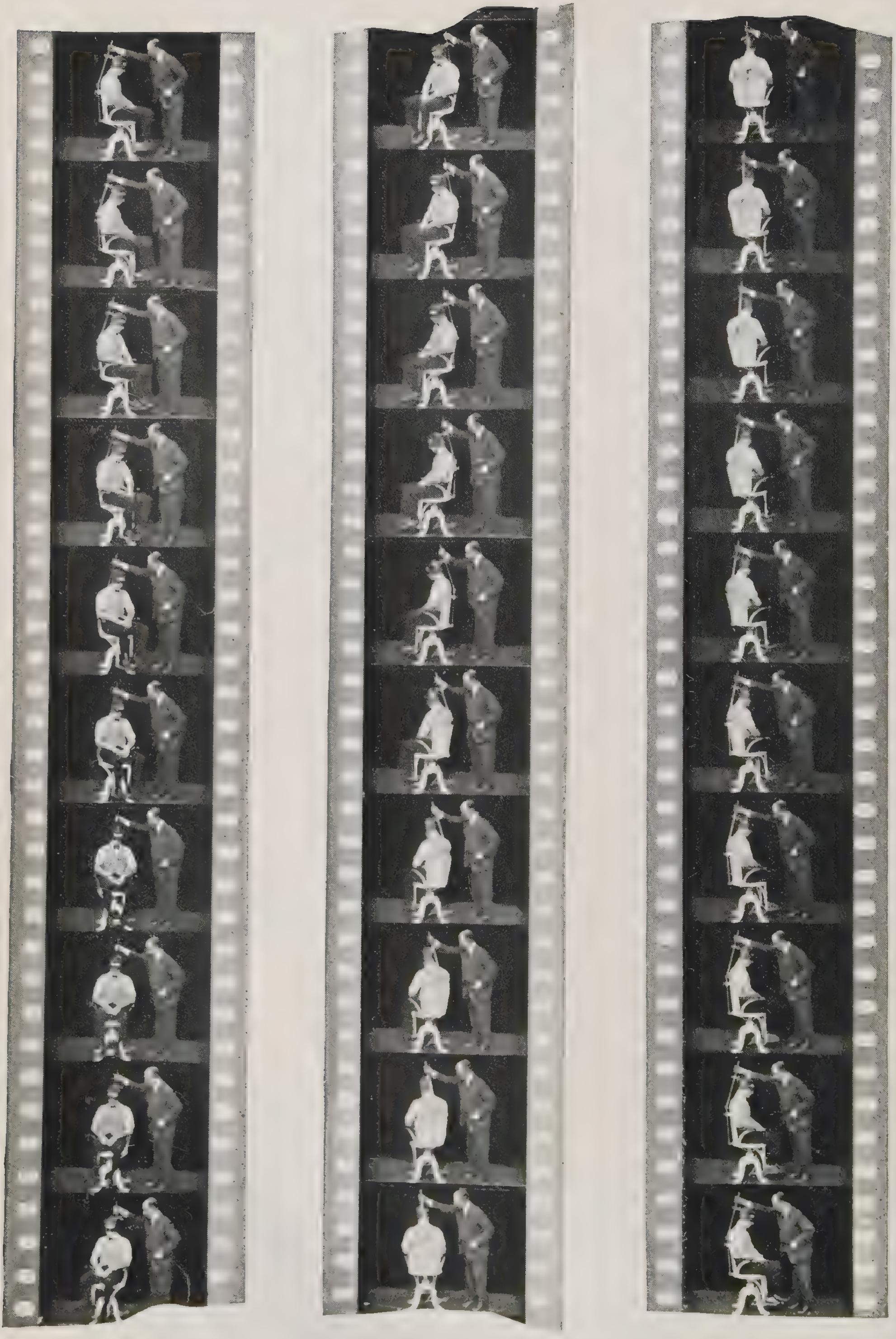

FIG. 1.— Turning to the right, head forward 30°, stimulating both horizontal semicircular canals.

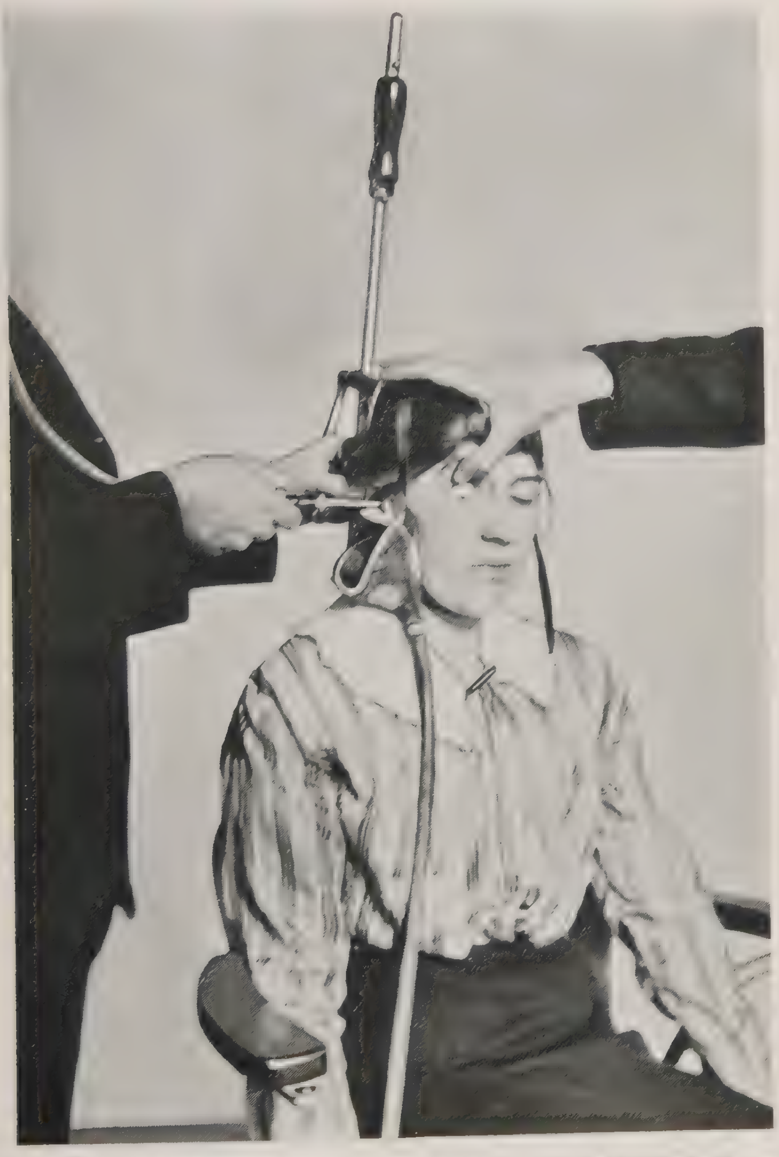

Fig. 2.—Method of [illegible]

left, a sensation of falling to the left, "past-pointing" to the right and actual falling to the right. Hot water produces exactly the opposite phenomena. This then, in brief, is the method of testing this *vestibular apparatus* or "Balance Mechanism."

The fundamental principles underlying these phenomena are very simple and may be expressed as follows: When "Father Adam" first turned to the right, to speak figuratively, the fluid (called "endolymph") in his semicircular canals lagged behind—relatively moved to the left. His sight and muscle-sense informed him that he was turning to the right; consequently he came to interpret endolymph-movement to the left to signify that he was turning to the right. He always turned *away from* the endolymph, and for this reason he recognized endolymph movement in one direction to signify that he was moving in the opposite direction. Each child born of Adam passes through the same experience—he learns to interpret impulses from the labyrinth just as he comes to interpret stimuli received from the retina. Images on the retina are upside down, but in the course of time the child learns to reverse this image in his consciousness and to realize that the external object is really right side up. Similarly through countless repetitions he comes to recognize that endolymph movement in one direction means that he is moving in the opposite direction.

When an individual moves past external objects, his *eyes* attempt to fix upon certain objects as they pass. He does this in the attempt to stabilize the sensorium. When traveling in a railroad train his eyes fix upon telegraph poles and other objects as they pass—consequently his eyes move in the direction of the passing object. Similarly, when he is turning to the right, his eyes, although closed, have been trained to move to the left in the direction of the external objects which are moving to the left.

Experimental stimulation of the ear, by turning in a revolving-chair or by douching with cold or hot water, produces an *artificial* movement of the endolymph. After an individual is turned to the right a sufficient number of times to cause the endolymph to catch up with the movement of the body, he feels that he is standing still, whereas he is actually turning to the right. As there is no movement of the endolymph in relation to the hair-cells within the ear, his interpretation is that he himself is not moving. Now if the chair be stopped, the endolymph by its momentum continues to move to the right; he therefore feels that he is rotating to the left, although he is actually sitting still. If the right ear be douched with cold water, the chilled endolymph moves downward to the right and the individual feels that he is falling to the left. Under all circumstances endolymph movement in one direction means to the individual that he is moving in the opposite direction; the eyes are always drawn in the direction of the seemingly passing object. This is the mechanism of the vertigo and the eye-movement caused by stimulation of the semicircular canals.

In the following chapters of Part I will be shown how the study of the balance apparatus, as just outlined, is raised out of the field of merely physiologic interest and how it serves a definite purpose in the "healing of the sick." It must be remembered above all that the work of otologists along the lines of the relation of the ear to the general system is only in its infancy, and in the following chapters we will attempt to give a hint of the *future* possibilities of this study as well as to indicate how the co-operation of otologists with those in other branches of medical work is already of use in general medical and surgical diagnosis.

## CHAPTER II

## THE EAR AND THE GENERAL PRACTITIONER

### Equilibration. The Study of Vertigo

The fact of paramount importance in the study of neuro-otology is that the ear is the most important organ in the maintenance of equilibration. Equilibration in general depends upon impulses from three sources—the kinetic-static sense, the sight and the muscle-sense. For centuries man has been credited with only five special senses. Within the last two decades the sixth sense, "Muscle, joint and splanchnic sense," came to be recognized. It is unlike the other senses in that it does not make its impression upon our consciousness. By means of this sixth sense the individual performs co-ordinate acts automatically and unconsciously. The recent studies of the internal ear show that the equilibratory portion of the ear constitutes a seventh sense, which may be termed the "kinetic-static sense." The kinetic-static function of the labyrinth is a separate sense just as truly as the sense of hearing or the sense of sight. Any special sense depends upon an end-organ for the reception of stimuli, nerves to convey these stimuli, and a nerve-centre to interpret their significance. The kinetic-static sense fulfils these requirements, as it is constructed on this plan, in that it consists of an end-organ, the semicircular canals and the vestibule, for receiving stimuli, a conducting nerve, the VIII Nerve, and definite tracts leading to brain-centres where the significance of the stimuli is interpreted in the form of equilibration, or that disturbance of equilibration which is known as vertigo.

It is therefore best to consider that there are seven special senses: (1) sight, (2) hearing, (3) taste, (4) smell, (5) touch, (6) muscle-sense, (7) kinetic-static sense.

Perfect equilibration is accomplished through an harmonious co-operation of the first, the sixth and the seventh senses—the eye, the muscle-sense and most particularly the kinetic-static sense.

This "sense trilogy" makes possible estimations of all sorts, orientation, locomotion, estimation of the rate of motion, estimation of weight, and realization of posture and position. Of the "trilogy," the kinetic-static sense is of peculiar importance, in that its "end-organ," the kinetic-static labyrinth, has for its *sole* function the maintenance of balance.

After impairment or loss of one of the senses responsible for equilibration, compensation may take place, to a certain extent. The tabetic may be taught to avail himself of his visual sense and of his static sense in co-ordinating his movements. Similarly, the blind man is able to stand or walk, until deprived of the guidance of either the muscle-sense or the kinetic-static sense. Deaf mutes, in whom the kinetic-static sense is destroyed, are enabled to maintain their balance by means of the sight and muscle-sense, and develop inco-ordination only in the dark or in the water. Full compensation, however, cannot take place unless two of these three senses remain unimpaired, and perversion of any one of them may be much more disturbing than its loss.

The realization that the ear is the organ of balance throws an entirely new light on the importance of this organ to the general practitioner. Previous to this conception of the ear as the essential organ of equilibration, the physician has regarded the ear somewhat as follows: if the patient is deaf a specialist should see what he can do to improve the hearing and if there is any inflammation of the ear it should receive local attention. Far more important and yet far less recognized and appreciated is the part that the equilibratory portion of the internal ear plays in the well-being of the entire body. A knowledge of this subject is necessary to the general practitioner because of

1. The value of the new ear-tests in *determining the cause of vertigo,* no matter what its type or origin; it is with such "dizzy cases" that the physician comes in daily contact; and

2. The necessity in his daily practice of recognizing pathologic conditions of the internal ear itself; such conditions are being continually overlooked at the present time because of a lack of familiarity with this new subject.

## VERTIGO

ALTHOUGH it has been generally recognized for many years that vertigo may result from ear disturbances, the conception that all vertigo, from whatsoever cause, is peculiarly an *ear study,* is the outcome of the study of neuro-otology. The general practitioner is constantly confronted with cases of "dizziness," and yet it is surely not overstating the fact to say that at best he can only guess at its clinical significance. Those suffering from it are at times so desperate that they would be willing to go to any length in order to obtain relief. The stability of the earth is one of the most fundamental facts of our experience—one of our essential concepts; it is no wonder that the ancients found it hard to believe that the earth moved. Therefore, if an earthquake shakes the very ground under his feet, man is astounded and feels that even the foundations of his reason and his hope for the future are being snatched away. The motion of the sea with its uncertainties and nauseating disturbances only occasionally is so overwhelming as to obliterate the assurance that this is but temporary and will be looked back upon with half-contemptuous self-confidence just as soon as one regains "terra firma." The sufferer from dizziness, however, has no such reassurance. "To him who wears shoes the whole world is covered with leather," and "He that is giddy thinks the world turns round." The dizzy man carries the terror of earthquake wherever he goes, feeling that for him all order of the universe is at an end; he is disabled for to-day and disheartened for the future. The inebriate suffers his loss of equilibrium and co-ordination under an exhilaration that robs it of its message to his benumbed brain; he is impressed with the seeming incongruity rather than with his own impotence. Normal equilibrium is essential not only for the worker but even to the idler if he is to live in comfort and safety.

Unfortunately in the past the physician could offer but little in the way of help. He has been accustomed to regard vertigo as something vague and mysterious and quite beyond the reach of medical aid, with the result that there is hardly any subject in the whole domain of medicine more chaotic than that of vertigo. Doctors repeatedly speak in a general and indefinite way of "intestinal" or "stomach" vertigo, dizzy spells from refractive errors, from Bright's disease, indigestion or neurasthenia, without thinking even for a moment of the real mechanism of its production. Above all, it has been impossible to know, in any given case, whether the vertigo was due to a functional or organic cause and still less to recognize whether the dizziness was of trivial significance or whether it was the forerunner of some grave affection.

In the light of the new ear-tests, vertigo should be regarded as a distinct clinical entity, deserving just as careful investigation and analysis as fever or any other distressing symptom. By vertigo is meant a subjective sensation of a disturbed relationship of one's own body to surrounding objects in space. It is not some general manifestation accompanying disorders of this, that or the other organ, but it is a disturbance perceived within a definite part of the brain, just as sight and hearing are perceived within the brain. Therefore, one should never speak of gastric vertigo, kidney vertigo, cardiovascular vertigo, idiopathic vertigo, or any other generalization which in its ultimate analysis means nothing. The stomach of itself, or the kidneys, or the heart, can no more produce vertigo than they can produce sensations of flashes of light, hallucinations of sound, or obsessions of smell. It is generally known and admitted as a matter of course, that the light, sound and smell-sensations in these instances are produced by irritation or stimulation within the brain of the visual, auditory or olfactory apparatus, as the case may be. All conscious sensations are cerebral. Headache, be it due to constipation, displacement of pelvic organs, or a gastro-intestinal disturbance, is nevertheless in the head. In just the same way, if a disturbance in any organ of the body is accompanied by vertigo, it is due to a direct attack on the apparatus capable of producing vertigo—namely, the internal ear or its intracranial distribution.

It must not be considered that vertigo can be caused only by a disturbance within the ear itself, for it is well known that various visual disturbances, cardiovascular affections, gastric or alimentary disorders also manifest vertigo as a symptom. The essential feature is that it is a direct action on the internal ears or their associated pathways in the brain, that is responsible for the vertigo. If the same pathologic cause, for one reason or another, fails to irritate the ear or its nerve-distribution, *there will be no vertigo.* The internal ears and the intracranial pathways constitute the apparatus that keeps us from "being dizzy." The vestibular mechanism accomplishes this by keeping the cerebrum continuously informed of our position in space and our relation to objects around us. In the presence, however, of some pathologic condition in the labyrinth or along any of its paths, a performance of this function is no longer possible; objects around us do not appear as they should; they either move or dance before us. Our own bodies no longer feel stable upon the ground—in other words, we are *dizzy* or experience sensations of vertigo which, if severe enough, result in our inability to move about or stand upright.

Viewed in this light many conditions in the category of the physician's daily experience—such as the symptoms of a "bilious attack"—attain a new significance. The mere ingestion of alcoholic beverages does not produce vertigo; it is only when the alcohol reaches the vestibular apparatus through the blood-stream that dizziness is produced. In consideration of these facts, to speak of the rôle that the ear and its ultimate tracts play in the production of vertigo, is like speaking of the rôle the heart plays in the production of cardiac murmurs. Therefore, just as we examine the heart and blood-pressure to determine the condition of the cardiovascular system, or as we test the urine in suspected cases of nephritis or diabetes, or as we have a Wassermann test made in cases suspected of syphilis, just so the ear-tests enable us to analyze the apparatus responsible for dizziness. Instead of guessing, we can then know the reason for the vertigo. We do not hesitate to say that it is very rare that a "dizzy" case remains obscure after these ear-tests and in most instances the diagnosis becomes clear and simple.

Vertigo is, therefore, essentially an *ear*-study. Have we in fact any other method of attacking this problem except by the ear-tests?

Surely any opinion based on history or analogy or empiricism is at best merely a conjecture.

We will attempt a classification of all possible causes of vertigo and then present actual cases of the various types, showing how the ear-tests have demonstrated the reason for the vertigo. If for any cause, immediate or remote, there occurs disturbance of the ear-mechanism, there is a resulting vertigo. Vertigo may be caused by

1. Involvement of the ear-mechanism by a lesion in the ear itself;
2. Involvement of the ear-mechanism by a lesion affecting the intracranial pathways from the ear;
3. Involvement of the ear-mechanism by ocular disturbance, either through the eye-muscle nuclei or through association fibres from the cuneus to the cortical terminus of the fibres from the ear in the posterior portion of the first temporal convolution;
4. Involvement of the ear-mechanism by cardiovascular disturbance; and
5. Involvement of the ear-mechanism by toxæmias from any organ or part of the body.

So far as we have been able to determine, this classification includes all the causes of vertigo; it is to be noticed that, regardless of the cause, the vertigo is due to an involvement of the ear-mechanism.

1. Lesions in the ear itself. A thorough knowledge of the recent advances in the study of the ear is of prime importance to the physician even from the standpoint of lesions of the ear itself. The general practitioner is familiar with conditions of the external ear, the middle ear and the *cochlear* portion of the internal ear. In the external ear, impacted cerumen, foreign bodies, eczema of the auditory canal, and furunculosis—all these conditions are obviously purely local and require local treatment. In the middle ear he encounters chronic catarrhal changes producing deafness, and various inflammatory conditions. To be sure, some of the inflammatory conditions of the middle ear have more than a local significance; abscess of the middle ear and mastoid empyema may

have far-reaching complications, such as meningitis, brain-abscess and lateral sinus thrombosis. Even in these conditions, however, the ear is after all merely the local source of infection. Affections of the cochlear portion of the internal ear are made manifest by impaired hearing and noises in the head; the attention of the physician, therefore, is immediately called to the ear; in fact the patient himself recognizes that he has an ear disturbance. A lesion of the vestibular portion of the labyrinth produces dizziness, staggering, nausea, vomiting, and perhaps diarrhœa, just as an affection of the cochlea produces noises in the head. When confronted with these symptoms, it is questionable whether at the present time the general practitioner is apt to suspect the ear as a possible source of all these phenomena. The external ear, the middle ear, and the cochlea, are of interest from a local standpoint, whereas the equilibratory portion of the ear is part and parcel and servant of the whole organism. A big, strong, healthy man is unable to stand or even to sit up in bed, because of a slight congestion of the internal ear: perhaps the pathologic process has produced a lesion no larger in extent than the head of a pin, yet the man is entirely incapacitated. Many such lesions endanger the life of the patient and yet may readily be overlooked unless the physician has come to recognize the need of ear-examination.

Non-inflammatory conditions of the inner ear may be produced by various toxæmias or by hyperæmia or anæmia of the labyrinth; these affections are productive of many annoying symptoms—deafness, tinnitus, vertigo and staggering—all bad enough in their way and also disabling, yet at the same time not serious in the sense of requiring immediate intervention of any kind. Not so with inflammatory conditions of the internal ear; here immediate expert attention is imperative and the patient's condition should be considered grave. Many unfortunate and at times tragic errors are made by physicians unacquainted with the significance of disturbances of this type.

Case 1.—John B., aged 46. August 3d, 1916, he felt a slight itching in the right ear and rubbed his ear with his finger. He was astonished and frightened to notice that everything in the room rotated rapidly. However, he paid no more attention to the matter. Two days later, while lying in bed on his back, he rolled over to the right side and was annoyed to find that the bed seemed to rise on the left side and he felt that he was falling out of the right side of the bed. He had always had a habit of shaking his head from side to side when saying "No" in conversation; he found that he had to stop this habit, as it caused vertigo. He then began to fear that "something must be wrong inside his brain." and consulted his family physician, Dr. Victor Janvier, who elicited a history of purulent discharge off and on from the right ear for over 40 years, following scarlet fever. Although there had been no discharge for the past four years, he recognized that the ear was probably the cause of all the symptoms. Ear-examination showed necrosis of the inner wall of the middle ear with a *fistula* leading into the horizontal semicircular canal. Local treatment has healed over this fistulous tract and has also apparently cured the purulent middle-ear condition. The man is not only entirely free from all attacks of vertigo, but is also cured of a condition which, if neglected, could have resulted in purulent labyrinthitis and meningitis.

Case 2.—George W., aged 30. Three weeks before admission to the hospital, while at work, was suddenly seized with dizziness, severe enough to make him fall over. He had headache, vomiting and diarrhœa and had to be taken home and put to bed. The physician consulted thought the man had "stomach trouble" and treated him accordingly. The vomiting had ceased, but as the vertigo and headache persisted, the patient left the doctor and applied to a medical dispensary for treatment. The physicians there thought his trouble was due to his eyes and referred him to the eye department. The ophthalmologists undertook to treat the case and prescribed a mydriatic. The patient was not getting better and consulted Dr. Shmookler, who found that there had been a chronic discharge from the right ear since childhood. He was convinced that the symptoms were referable to the ear and therefore sent the patient to the hospital again, but this time to the ear-service. The ear-examination disclosed a circumscribed labyrinthitis—a beginning extension into the internal ear of the purulent process in the middle ear—a serious condition calling for immediate local care of the ear.

Case 3.—Maurice B., aged 51, was seized with dizziness, staggering, vomiting and diarrhœa on board ship, coming back from Europe. The ship's doctor naturally attributed it to seasickness, but after the patient reached land the symptoms did not disappear. A few weeks later the vertigo and staggering were so marked that he could not stand nor even sit up in bed. Dr. Kercher, his physician, immediately suspected that it was an ear-condition, especially as the patient had had running ears off and on nearly all his life. On examination we found that douching the left ear failed to produce the normal responses from the semicircular canals, namely, nystagmus, vertigo, past-pointing and falling. This demonstrated an involvement of the horizontal canal and also of the vertical canals, whereas, although the hearing was markedly impaired, the *cochlea* was entirely normal. This apparently surprising phenomenon was easily explained as due to an extension of the inflammation from the middle ear through the oval window, involving the end-organs of the semicircular canals, but so limited that it did not extend forward into the cochlea. Rest in bed and local treatment of the ear was followed by uninterrupted convalescence. A mastoid operation should now be done to cure the purulent middle-ear condition and so prevent a recurrence of such an attack. In this case, according to old standards, with a perfectly functionating cochlea, not the slightest earache, no discharge from the ear or lighting up of an old middle ear-suppuration, and with the patient himself not complaining of his ear nor calling attention to it, there was nothing whatever to suggest ear-involvement. He simply presented a picture of a big, healthy man unable to stand up because he was dizzy. It was only this doctor's familiarity with the significance of dizziness and staggering that made him think of the ear.

Case 4.—John R., aged 58. Attacks of vertigo for 15 years at intervals of a year or six months. At no time during these attacks did he notice any tinnitus. Occasionally when attending to his regular business he felt that he was about to fall; this sensation passed off in a few minutes. Examination showed entirely normal internal ears and all intracranial pathways from the ears. Local treatment of the left middle ear, which had been the seat of occasional discharge, completely cured his attacks of vertigo and he has had no further attacks during a period of three years.

Summarizing, inflammatory conditions of the middle ear, such as an acute or chronic otitis media may produce only irritative effects upon the labyrinth, so that the patient suffers from more or less vertigo only so long as the acute stage of the congestion lasts; the vertigo vanishes with the disappearance of the inflammation. Slow degenerative changes within the labyrinth similarly produce "attacks" of vertigo from time to time. On the other hand, sudden destruction of the whole or part of one labyrinth is accompanied by profound vertigo, nausea, vomiting and loss of equilibration. Such destruction of the whole or part of the labyrinth may be produced rarely by trauma, but usually by hemorrhage or serous effusion into it, and may occur in diabetes, nephritis or in any condition in which the vascular system is affected. It is this class of cases that exhibits the so-called "Meniere's symptom-complex." The hearing in these cases is usually markedly affected if not altogether gone, and the condition is characterized by an attack of sudden onset, the violence of the symptoms quickly reaching a climax and then gradually subsiding, and all of the symptoms disappearing when the brain-centres have learned to compensate.

When a patient complains of dizziness and staggering, with or without nausea and vomiting, one of the first thoughts, therefore, should be of a possible lesion of the ear itself.

2. Lesions within the brain, causing vertigo by affecting the intracranial pathways from the ear, such as tumor, hemorrhage, thrombosis, infarct, abscess, gumma, tubercle, leukæmic infiltration, specific neuritis, multiple sclerosis, syringomyelia, polio-encephalitis or meningitis.

The value of ear-examination in the differential diagnosis between lesions in the ear and lesions in the brain is best illustrated by citing two cases. In one case the history and symptoms indicated a lesion of the ear, and yet it turned out to be an intracranial lesion; in the other case, the history and symptoms indicated an intracranial lesion and yet it turned out to be a lesion in the ear.

Case 5.—J. B. K., aged 39. This man, in September, 1914, while stepping off a train, was suddenly seized with violent vertigo, nausea and vomiting. The vertigo was so severe that he fell to the platform, and was taken to the hospital where it was necessary to keep him in bed for ten days; during this time the vertigo gradually diminished. The patient also had complete deafness in the right ear. Here we have the typical classical picture of the so-called "Meniere's disease"—a sudden hemorrhage into the internal ear. Yet it turned out to be a beginning tumor in the right cerebello-pontile angle from which the patient died in seven months.

Case 6.—Mrs. Agnes G., aged 44. July, 1914, the patient suddenly began to have difficulty in walking and in September had severe attacks of vertigo, nausea and projectile vomiting. The diagnosis of brain-tumor was made and neurologic consultants found that the symptoms and tests suggested that the tumor was in the right cerebellar hemisphere. This diagnosis was confirmed by the X-ray report, which stated that it was a cyst in the right cerebellar hemisphere. The surgeon was about to operate, but hesitated because the ear-examination suggested that the lesion was in the right labyrinth and that the cerebellum appeared normal. The past-pointing after ear-stimulation was normal—both arms past-pointed properly both to the right and to the left. The patient had an uninterrupted convalescence without operation, has given birth to a healthy child and has remained in perfect health ever since—two and one-half years.

3. Ocular conditions producing vertigo are naturally best studied by the ophthalmologist. If the ear-tests fail to show any impairment of the ears or their intracranial pathways, an eye-examination is indicated. Many cases of vertigo are cured by correction of ocular defects. Many ophthalmologists have been so impressed with the number of cases of vertigo cured by correction of ocular defects, that they regard the eye as the most important organ in

the causation of vertigo. The eye, however, is only a contributing cause of vertigo in certain cases. The eye is not the organ of balance. If an eye is removed, blindness results, but no vertigo; on the other hand, if the internal ear is destroyed, or for that matter only slightly irritated, there immediately result vertigo and loss of equilibration. It is only when an ocular defect as in muscle-paresis affects the ear-mechanism that vertigo results.

4. Involvement of the ear-mechanism by cardiovascular disturbance. This classification includes all cardiovascular conditions which produce either congestion or ischæmia within the cranium. If, as in shock, there occurs a cerebral ischæmia, the temporarily poor blood-supply to the psychic centres causes faintness and perhaps unconsciousness; similarly, the poor blood-supply to the ear-mechanism, either peripheral or central, produces vertigo.

5. Toxæmias affecting the ear-mechanism; these include ptomaine poisoning, alcoholism, poisoning by chemicals such as lead, quinine, salicylates of soda; nephritis, gout, rheumatism, syphilis and the toxæmia of infectious fevers such as mumps, scarlatina and typhoid fever. These toxæmias may be grouped into two classes.

(*a*) Evanescent toxæmias which have produced no degeneration of the cellular elements within the internal ear or its intracranial pathways.

(*b*) Toxæmias which have produced a definite impairment of some portion of the ear or its pathways.

(*a*) The cases of evanescent toxæmia constitute a very large proportion of the cases of vertigo that are seen by the physician. The simplest illustration of this type of vertigo is the dizziness produced by the ingestion of alcohol. As stated previously, the mere presence of alcohol in the stomach does not produce vertigo; it is only when, through the blood-stream, the alcohol reaches the ears and the brain that the individual becomes dizzy.

Case 7.—Mrs. Van S., aged 32. This patient had no symptoms referable to her ears, but was referred by Dr. John McGlinn because she complained of vertigo. In October, 1913, she awoke in the morning with a violent attack of vertigo, accompanied by nausea, retching and diarrhœa. The attack suggested ptomaine-poisoning in every particular, except that there was no pain in the abdomen. These symptoms continued for 40 hours, and then the patient improved progressively for the following four days. On the fourth day she was out of bed and apparently had entirely recovered; but on bending over to kiss her boy she suddenly felt that she was plunging forward through a door. This second attack lasted for another day. Gradual improvement followed, so that at the time of the ear-examination, ten months later, she stated that she noticed vertigo only when she lay down at night. On October, 1914, she awoke in the morning with the sensation that the bed was falling over; this attack, like the others, gradually diminished in severity after a few days. Between the attacks she merely notices that when she goes to bed at night it takes her about half an hour to go to sleep because she feels a little dizzy, and that she cannot sew or read because it makes her dizzy. Examination of the ears showed normal cochleas, normal semicircular canals and normal pathways from the ear throughout the brain. The diagnosis suggested under these circumstances was that we were dealing with a purely functional neurosis, or with a mild toxic irritation of the internal ears. On receiving this report, Dr. McGlinn recognized that the probable source of this toxæmia was a pyurea due to a pyelitis and an associated cystitis. Under the vigorous use of urotropin, the pyurea was markedly improved. Dr. McGlinn noted distinctly that when the pyurea was most marked, her vertiginous attacks were at the highest and that when the pyurea was least marked there was practically no vertigo. During the past year and a half she has been practically free from either the pyurea or the vertigo. In the past few days, however, the pyurea has returned, and with it the vertigo.

(*b*) Toxæmias which produce a definite impairment of the internal ears include the powerful toxins such as in mumps or syphilis, and also the repeated assaults of a milder toxin such as those from the gastro-intestinal tract or from a focal infection.

Case 8.—Mrs. W., aged 58. The patient's first attack of vertigo occurred in 1897; it lasted only a few minutes; she had three or four attacks during the next 14 years. A severe attack in 1911 occurred while she was walking along the street; these attacks came on at irregular intervals. In October, 1915, a severe attack occurred while she was sitting in the theatre and she was taken to the hospital where she remained for seven days. Because of the attacks of nausea, vomiting, diarrhœa and emaciation, a diagnosis was made of gall-bladder lesion; operation, however, showed the gall-bladder to be negative. Subsequent ear-examination made it perfectly simple that all her symptoms had been due to recurrent attacks of labyrinthine irritation. Her physician, Dr. R. L. Pitfield, has noted that when she eats fish or eggs she has a marked gastro-intestinal disturbance and that vertigo accompanies this disturbance. Under dietetic treatment she has recovered and at the present time is in excellent health.

Case 9.—Miss C. H., age 27. This patient was referred by Dr. Francis Packard, who felt it advisable to remove the pathologic tonsils, but hesitated to do so because the patient complained of severe and continuous vertigo for a period of over six months. This dizziness was almost continuously present; it made no difference whether she was lying down or standing. Occasionally at night the vertigo would be so severe as to wake her from a sound sleep. For the previous year or two, the patient noticed that when going up or down in an elevator she would almost faint; six months ago, however, the definite vertigo began and there appeared at the same time "an appearance of an eruption just under the skin." She has always had exceptionally good health until this skin-eruption and vertigo commenced. The report of the ear-examination read: "There is no evidence of any intracranial involvement along any of the pathways from the ear. There is a proportionate impairment of nystagmus, vertigo and past-pointing, indicating a slight impairment of function of the vestibular portion of the right labyrinth. The diagnosis suggested is a toxæmia which has affected the internal ears at intervals, thus producing the vertigo; the condition is in no sense serious. If the toxæmia can be eliminated it is possible she will have no more attacks of vertigo." Dr. Packard removed the tonsils; in a few days there was a complete disappearance of the vertigo and the skin eruption also disappeared at the same time.

Case 10.—Hugh H., aged 42. The patient was referred by Dr. Vincent Lyon with a history of an attack nine months before of acute articular rheumatism involving almost all the joints. At the time the patient was confined to the hospital for 17 days and after leaving the hospital continued to have occasional recurrence of the involvement of the joints. Three months after the original attack, the patient began to complain of vertigo and staggering and was referred for an examination of the ear in the attempt to discover the cause of the vertigo. The ear report read "Examination shows a distinct impairment of both internal ears, in both the cochlear and vestibular portions. This suggests that a source of toxæmia must be searched for. It may be that the same toxic substance that causes his joint involvement has also caused this toxic labyrinthitis." X-ray showed abscesses at the roots of two teeth; these abscesses were drained by the removal of these two teeth. Within a week the patient ceased to have any vertigo or staggering, and also all the rheumatic symptoms entirely disappeared. That the removal of the focal infection at the roots of the teeth cured this patient entirely of all his symptoms makes it strongly suggestive that this focal infection was responsible for his entire illness; the toxæmia on the one hand had involved the joints and on the other hand had involved the internal ears, thus producing the vertigo and staggering.

Summarizing therefore:

1. Vertigo from whatever cause is a disturbance of the vestibular apparatus, which we have spoken of as the ear-mechanism.

2. Disturbances of the vestibular apparatus can be definitely analyzed by means of the ear-tests.

3. Cases of vertigo need no longer be regarded as vague or mysterious but may now be cleared up by means of the ear-tests. Vertigo, therefore, need no longer be a term wherein to cloak

our ignorance as to the many diverse conditions of which it is a symptom.

In any given case of vertigo the *first thing* to be done is to examine this ear-mechanism that is responsible for the vertigo. The ear-tests will always show either abnormal or normal responses; if the responses are abnormal, the tests will help to determine a lesion either within the ear or within the brain. If the responses are normal we have then narrowed the diagnosis down to (1) a purely functional neurosis, (2) an ocular disturbance, or to an evanescent toxæmia the source of which must then be looked for. The ear-examination, to be sure, does not determine everything whatsoever that has to do with vertigo, but it certainly brings order out of chaos in these cases and makes possible accurate diagnosis and intelligent treatment. Vertigo, therefore, is essentially an ear study.

The methods of carrying out the ear-tests are described in Part II of this book.

# CHAPTER III

## THE EAR AND AVIATION

At the present hour perhaps the most valuable service that the otologist can render to the Government is in the Aviation Service. In aviation we have a practical example of the importance of the ear in maintaining equilibrium. It has been noted in the previous chapter that after the loss or impairment of one of the three senses responsible for equilibrium, compensation may take place to a certain extent. It is not necessary for an individual to have a perfect muscle-sense, perfect sight, and perfect internal ears in order to have fairly good equilibrium; this, however, takes for granted that the individual is on ''terra firma.'' When the human being becomes a bird, as it were, he suddenly finds himself in an entirely new environment. Without functionating internal ears it is impossible for an individual to be a good bird-man. When flying through the air, on what does the aviator rely in order to maintain his equilibrium and that of the aeroplane? Can he rely on sight? Hardly, for when he is sailing through the clouds or darkness, his eyes cannot give him the slightest information about his position in space—not even whether he is right side up or upside down. As regards the muscle-sense, it is undoubtedly true that it plays a certain part; but when the aviator is seated on an unstable and rapidly moving machine, it is hardly conceivable that the weight of his body could determine and maintain his position in space merely by the sensing of gravity. In order, therefore, to preserve that wonderful accuracy necessary in controlling such a delicate mechanism as the flying machine, he relies pre-eminently on his ear balance-sense. It is easily conceivable that some of the unexplained accidents in aviating may be due to a concussion of the internal ear produced either by the deafening roar of the engine or by the decrease of the air-pressure when at great heights. Also in a rapid ascent from a denser to a rarer air there occurs an oxygen insufficiency which has a direct effect upon the ear-mechanism through the blood-stream. It is also highly probable that many an aviator has gone to his death because, all unknown to him, he did not possess a normal ear-mechanism; in the presence of a combination of difficulties in which all normal faculties would be requisite, he was unable to maintain his balance because of an imperfect ear-mechanism.

To realize the importance of the ear in the matter of flying, it is only necessary to consider a bird flying in a cloud. His muscle-sense naturally means nothing to him; his sight is of no help. He relies exclusively, therefore, upon normal functionating semicircular canals, which we know are wonderfully well developed in the bird.

Summarizing, therefore, even when an individual is standing or walking on the earth, his ears constitute his sense-organs of balance; as long as he remains upon the earth he has in addition the contributory help of information received from his muscle-sense and his sight. When he rises above the earth and flies in the dark it is obvious that these contributory factors are practically eliminated and he must rely almost exclusively upon the ear balance-sense.

Since normal internal ears are such an important asset—in fact a prime requisite—for the aviator, common prudence would suggest a most careful examination of the degree of function of one's internal ears before taking up flying as an occupation. The ear-tests furnish exact and mathematical data concerning not only the function of the internal ear, but also of the entire vestibular apparatus; this includes, besides the ears themselves, the VIII Nerve, the brain-stem, the cerebellum and the entire balance-mechanism.

When it was announced that a state of war existed between the United States and Germany, it at once became apparent that a tremendous number of aviators must be secured for the military service within the shortest possible space of time. The medical problem consisted of selecting thousands of physically equipped candidates for aviation and placing them in training for war-service immediately. The medical department found it necessary to decide upon new methods of physical examination and to adopt new standards of physical qualifications for this branch of the service. This had to be done not merely for one place or for one examining group; it was necessary to make the tests practicable for cities in all parts of the United States, without, at the same time, in any way lowering the requisite rigid standards or lessening the completeness of the examination. How could this be done? In a word, such an ideal could be attained only by (1) the standardization of the *tests,* and (2) the standardization of the *examiners.*

The methods presented in this book were adopted in May, 1917, as the standard for the U. S. Army. The following briefly summarizes the fundamental principles underlying the examination for aviators. The aviator must be in a sense the "superman." He must have 20/20ths vision without glasses. He must have 40/40ths hearing. Such requirements are higher than for most branches of military service. A candidate for every branch of service must conform to a certain physical standard; he must be a normal man, such as would satisfy the average life insurance company, with the additional requirements of a certain relative height, weight and chest measurement and a definite visual and auditory acuity. Like any other candidate for service, the aviator must conform to all these requirements and in addition must possess visual and auditory acuity to a high degree. There is, however, an attribute not required by any other branch of the military service which is indispensable to the perfect aviator—a good *balance-mechanism.* Therefore the peculiar test, applied to the aviator alone, is the special examination of the equilibratory portion of the internal ear.

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### PHYSICAL EXAMINATION OF APPLICANTS FOR DETAIL IN THE AVIATION SECTION, SIGNAL CORPS, U. S. ARMY

### EQUILIBRIUM (VESTIBULAR TESTS)

The nystagmus, past-pointing and falling after turning, are tested. The turning-chair must have a head-rest which will hold the head 30° forward, a foot-rest and a stop-pedal. (The chair shown on page 235 is officially required. This made possible the establishment of an absolute standard. While the tests could be made by using other types of turning-chair, an exact quantitative estimation of the responses can be definitely established only by the use of a standardized chair.)

(*a*) *Nystagmus.* First of all a spontaneous nystagmus must be looked for. It is noted whether there is any twitching of the eyes when gazing straight ahead, or looking either to the extreme right, the extreme left, up or down. Head forward 30°; turn candidate to the right, eyes closed, 10 turns in 20 seconds. The instant the chair is stopped, click the stop-watch; candidate opens his eyes and looks straight ahead at some distant point. There should occur a horizontal nystagmus to the left of 26 seconds' duration. Candidate then closes his eyes and is turned to the left; there should occur a horizontal nystagmus to the right of 26 seconds' duration. A variation of 10 seconds is allowable (either as low as 16 seconds or as high as 36 seconds).

(*b*) *Pointing:*

1. Candidate closes eyes, sitting in chair facing examiner, touches examiner's finger held in front of him, raises his arm to the perpendicular position, lowers the arm and attempts to find the examiner's finger. First the right arm, then the left arm. The normal is always able to find the finger.

2. The pointing test is repeated after turning to the right, 10 turns in 10 seconds. During the last turn, the stop-pedal is released and as the chair comes into position, it becomes locked. The right arm is tested, then the left, then the right, then the left, until candidate ceases to past-point. The absolutely normal will past-point to the right 3 times with each arm, if needless delay is avoided. (However, one past-pointing to the right of each arm qualifies, if the nystagmus and falling are normal.)

3. Repeat pointing-test after turning to the left. (Similarly one past-pointing of each arm to the left qualifies, if the nystagmus and falling are normal.)

(*c*) *Falling.* Candidate's head is inclined 120° forward, eyes closed. Turn to the right, 5 turns in 10 seconds. On stopping, candidate quietly sits up and should fall to right. This tests the vertical semicircular canals. Turn to the left, head forward 120°; on stopping, the candidate again sits up and should fall to the left.

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Obviously these tests as presented in this blank are not intended to make a diagnosis of a pathologic lesion. The object is merely to determine whether or not the ear-mechanism is normal. If, in these tests, the candidate shows normal responses in nystagmus, past-pointing and falling, he is fit for the aviation service; if he does not, he is unfit for that service. These simple turning tests eliminate all unnecessary, time-consuming, diagnostic procedures. The entire series of tests as outlined in the blank requires only 3¾ minutes; and yet in this short time we are able to determine the integrity of the internal ears, the VIII Nerves, and the pathways through the medulla oblongata, the pons, the six cerebellar peduncles, the cerebellum itself and the cerebral crura to the cerebral cortex—the "balance-mechanism."

Incidentally, these tests are in no sense severe and are in fact seldom regarded even as unpleasant. Occasionally nausea occurs after a few turnings; it is then merely necessary to stop the examination for the time being and to complete the remainder of the tests after an interval of a half-hour. There is no need whatever to make these tests in any way distressing to the candidate.

These turning-tests quickly separate the obviously fit from the unfit. The majority of the candidates show normal responses; no further testing is required and they therefore qualify and are accepted. Some candidates show such markedly subnormal responses that they are immediately disqualified and rejected. A limited number give what might be termed "border-line" responses; the question then arises, has this particular applicant *sufficient* balance-sense to become an aviator? It is here that the caloric test is useful. The turning has tested both the right and left ears simultaneously. The caloric method enables us to test each ear separately. Water at 68° F. is allowed to run into the external auditory canal from a height of about 3 feet through a stop-nozzle, with the head tilted 30° forward, until the eyes are seen to jerk or the individual becomes dizzy. The length of time from the beginning of the douching until the jerking of the eyes becomes apparent, or until the applicant says he is dizzy, is accurately measured by a stop-watch. The type of nystagmus is then noted. It should be rotary and the direction of the jerk should be to the side opposite the ear douched. The length of time shown by the stop-watch in the normal is 40 seconds. The eyes are then closed and the past-pointing is taken. The head is then immediately inclined backward 60° from the perpendicular (or 90° from the original position). There should then appear a *horizontal* nystagmus to the side opposite to the ear douched. The eyes are then closed and the past-pointing is taken with the head in this position. The left ear is then douched and the same procedure carried out. If the caloric test applied to one of these "borderline" cases shows only a slight impairment of the responses from each ear, the candidate is qualified. A slight impairment would be indicated if instead of the normal 40 seconds of douching, there was required not more than 90 seconds of douching. If one ear shows normal responses, whereas the other ear shows responses only after more than 90 seconds of douching, the candidate is disqualified. Care should be taken to be certain that the cold water is reaching the drum-head during this caloric test, as wax or other obstruction in the external canal would interfere with the responses in a perfectly normal individual.

The examination of a candidate for the aviation service is a different matter from the examination of a patient. We are dealing with an alert mind, anxious to make the best showing possible. Many candidates feel that "jumping eyes" and evidences of vertigo are signs of weakness and would be counted against them. Because of this anxiety, many candidates attempt to shorten the duration of the nystagmus by fixing the eyes on some near object. It is perfectly possible by fixing of the eyes to shorten the duration of the nystagmus. It is a common experience in this work to note that when many observers are standing about the candidate and in his line of vision that the nystagmus is usually of short duration. If these observers are asked to stand back out of the line of vision, the same candidate almost invariably shows a longer duration of nystagmus. Further to obviate fixation of the eyes, it is always necessary to place the chair near a large window so that the candidate has an unobstructed view of an object placed at "infinity"—over 20 feet. Furthermore, instead of obeying the natural impulse to past-point, they frequently make a mental calculation as to the vertigo induced and voluntarily attempt *not* to past-point. This is also true of falling. In such instances, the examiner, bearing in mind the type of individual with whom he is dealing, urges the applicant to "act perfectly naturally," and is then usually able to elicit a true response to the test. Supposing, however, that the candidate still fails to past-point, although he has shown a normal nystagmus and falling —we are able to decide the matter finally in the following way: First, the quantitative estimation of vertigo is taken. The after-turning vertigo is measured as follows: Candidate is turned to the right, 10 turns in 10 seconds, with eyes closed, as follows: As he is being turned, he is asked to describe his sensations and to keep on telling in which direction he feels he is being turned; thus he will say "to the right, to the right, to the right," etc., until the chair is stopped, then he will feel himself turning in the opposite direction and will say, "Now I am going to the left, to the left, to the left," etc., when as a matter of fact he is sitting perfectly still in the chair. The duration of this sensation of vertigo from the time that the chair is stopped until he ceases to feel that he is going to the left is taken in seconds with the stop-watch. The normal should show an after-turning vertigo of 26 seconds. The test is then repeated by turning to the left and the candidate should exhibit a vertigo in the opposite direction of 26 seconds. Should the candidate show over 16 seconds of vertigo in both directions, having previously shown a normal nystagmus and falling, the examiner then realizes that the absence of past-pointing was probably due to a calculated correction rather than to any pathologic condition. The question is then definitely determined by douching the ears. Although a candidate can estimate the significance of the sensation of vertigo after turning, he has no control over this sensation after douching; he is unable to calculate the meaning of the vertigo produced by the caloric test. Therefore, if he fails to past-point after the douching-tests, he is definitely disqualified.

So much for the standardization of the tests themselves. Equally important was the problem of the standardization of the examiners. For this purpose a medical officer was sent under special orders to each of 35 cities throughout the United States and in each one of these cities there was established a medical unit for the examination of candidates for the aviation service. The requirements of the tests were fully explained to each medical unit, so that not only the same equipment is used, but also exactly the same technic. This rendered it impossible for any candidate to say, "I wish I had been examined in a certain city where the tests are easy, rather than in a certain other city where the tests are exacting." Those otologists were selected who were most expert in the Barany tests and they were given intensive training by the medical officer sent for the purpose of establishing a uniform technic. Thus in a few months the testing was put on an absolutely uniform basis in all examining centres.

In order to save time, already existing institutions, such as large hospitals or State Universities, with their equipments, were utilized as these examining centres. Volunteer staffs of civilian consultants were locally organized and the work of the examining centres systematized to a point of highest efficiency, with the result that within a few days of the arrival of the medical officer at the examining centre the work was in full swing. By this method of decentralization, the examination of thousands of applicants in a minimum space of time was made possible. First of all it was necessary to make sure that those in charge of each examining centre were fully equipped and capable to make these tests; this once assured, full authority and responsibility was vested in the medical officer in charge of each centre. Thirty-five medical aviation centres, each examining from ten to forty candidates a day, provided immediately the thousands of men required.

It is obvious to the candidate himself, that if he is deficient in his ear balance-sense he is not only a danger to the Service.

but he is also unnecessarily imperiling his own life when he attempts to fly. Certain members of the medical profession and certain veteran fliers, because of the newness of these methods, expressed skepticism in regard to their value. One physician, a member of an examining unit, was surprised to find that when he himself was examined, he showed an almost complete lack of ear balance-sense. The turning and caloric tests failed to produce nystagmus, vertigo, past-pointing and falling. This impairment of the ear-mechanism was directly traceable to a severe attack of mumps in childhood, during which he had suffered from both of the usual complications of mumps—an orchitis and an involvement of the internal ears. His skepticism was changed to enthusiastic conviction when he thus was made to realize that without the Barany tests he would have had no conception of this physical disability, the only suggestion of which, up to that time, had been his knowledge that he was unable to become seasick.

All experienced aviators that have been examined have, without exception, shown normal responses in the turning-chair. Those veteran aviators whose attention has been called to these methods of testing, have expressed their conviction of the practicability of the safeguards provided by these tests in determining whether or not a man has what they speak of as "air-sense." One seasoned American flier, of 15 years' experience, stated that his doubt was changed to conviction after he was examined in the turning-chair; he volunteered a statement that from his intimate knowledge of the circumstances attending the death of at least three aviators, who were killed while flying, their failure to negotiate difficulties of no unusual degree which resulted in their death, might now be understood as an expression of their lack of this sense. He further added, that in his judgment, if these tests had been available in the early days of flying, many lives might have been spared.

The value of the past-pointing test is well illustrated by one candidate who proved to have a post-traumatic cerebellar lesion, which was first detected by the isolated absence of past-pointing of the left arm to the right. Neurologic examination then demonstrated classical signs of lesion of the left cerebellar hemisphere.

Here was a man with distinctly impaired cerebellar function attempting to enter a service in which the utmost demands are made upon the cerebellum.

One ardent applicant was disqualified because of a markedly impaired internal ear, in both the cochlear and vestibular portions. It has since been learned that he went to another country where he was accepted in the aviation service as a flier. Obviously the requirement in that country is not so high as in the United States. At the present time in the United States a high standard is required because this country is in a position to pick and choose. The vast numbers of splendid applicants makes this possible. For this reason these hand-picked men will constitute not only the largest but the most capable and finest aviation service in the world.

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## CHAPTER IV

## THE EAR AND SEASICKNESS

"MAL-DE-MER" is a phenomenon resulting from movement of the endolymph within the internal ear. The history of seasickness dates as far back as man's first attempts to brave the dangers of the sea. It is difficult to understand why the ease with which one succumbs to seasickness should always have been a subject of ridicule, but the literature of all nations abounds with gibes and jests about the sufferer from *mal-de-mer*. Those who have been subjected to it, however, can state with a good deal of assurance that it is distinctly a serious matter, deserving as much sympathetic consideration as any other distressing ailment.

Theories as to the cause of this malady and remedies suggested for its relief have been innumerable and with the advent of each new explanation would come a host of remedies for its prevention and cure. A perusal of the literature on the subject, which is enormous, shows that every drug in the pharmacopœia has been considered useful; this of itself signifies that nothing really useful had been discovered. The medical profession, appealed to from time immemorial by a suffering traveling public, could afford no relief; so it is no wonder that even today thousands of people would not think of going to sea without protecting themselves, as they think, with some remedy suggested by some friend who has considered it useful, such as a belladonna plaster, a sheet of foolscap paper, or other applications over the *abdomen.*

Without a knowledge of the true etiology of this condition, the only treatment that appeared at all rational was the symptomatic; as nausea and vomiting are the most evident and predominant symptoms, everything centred about remedies which were supposed to relieve these symptoms. It is of course easy to see why there should have been the gastric theory of the causation of this malady; nausea and vomiting naturally suggested a "bad stomach." Based upon this theory, some were advised not to eat at all or only very little, whereas others were advised to indulge in a liberal diet. This theory, however, has come to be disregarded; any observing clinician could no more ascribe the nausea and vomiting of *mal-de-mer* to a gastric affection than he could ascribe the nausea and vomiting associated with brain tumor or meningitis to a disturbance in the stomach.

The *optic* theory was in vogue for a considerable time and had many adherents. According to this explanation, individuals were seasick because of the ever-changing images on the line of the horizon and of the swaying decks and spars. The observation that the administration of atropin seemed to be helpful appeared to lend considerable credence to this view. For a while atropin-blinded individuals, wearing violet-tinted glasses, were quite numerous aboard every ocean-bound vessel. The fact, however, that seasickness did not spare individuals who were asleep in their darkened berths soon furnished an insuperable obstacle to the correctness of this theory; and it soon lost its hold upon the traveling public.

The flushed appearance of an individual prior to succumbing to the malady suggested to some that a *cerebral congestion* was the basic underlying factor; on the other hand, the anæmic pallor gave origin to a theory of cerebral *ischæmia.* One author announced a positive cure for seasickness without the use of drugs, laying the blame for seasickness on the maintaining of a tense musculature while on board ship and suggested that all the individual need do is to relax completely and to assume the swaggering gait of the hardened sea-dog to do away forever with the discomfort and terrors of *mal-de-mer.* Perhaps the most vague of all suggestions, propounded in all seriousness, is that seasickness is due to a ''miasmatic intoxication.''

In this manner theory followed upon theory and ''cure'' upon ''cure.'' Vasomotor stimulants and vasomotor depressants; nerve excitants and nerve sedatives; gastric digestants and local anæsthetics—all have been tried only to be discarded.

With the discovery of the physiology of the kinetic-static labyrinth and a study of the phenomena attendant upon the stimulation of this organ, the true etiology of seasickness at last appeared. Many authors, among them Neumann, Barany, Ruttin, Spira, Champeaux, Trotsenburg, Minor, Savory and Barnett, have written excellent scientific and reasonable treatises on this subject, so that it is rather surprising that a knowledge of the etiology of this condition is not universal; but as Barany very clearly points out, the present chaotic views among not only the laity but even the medical profession in regard to the nature and causation of seasickness must be ascribed to the undeniable fact that the mechanism of the internal ear is not yet generally understood and appreciated.

The *symptoms* of *mal-de-mer* as observed and described by many authors are fairly constant. There is first noticed a more or less marked dizziness which may persist for a considerable time without any other symptom. Accompanying this vertiginous feeling is a staggering gait when motion is attempted. The early warm flushing of the face becomes quickly replaced by a feeling of intense chilliness accompanied by pallor and cool clammy skin with profuse perspiration. Nausea makes its appearance early but is not quickly followed by vomiting; when the vomiting finally does appear there is occasionally a marked feeling of relief and with some individuals the attack ceases there and then. This, however, is the exception; as a rule the respite is of but short duration and the nausea and other disagreeable symptoms soon recur. The vomiting is particularly distressing; continuous and painful retchings continue until nothing is brought forth but greenish bile-stained mucus. Some patients practically go into a collapse and remain in a state of shock as long as the voyage lasts, while others have varying intervals of comparative comfort.

It is now universally agreed that these symptoms are brought on by the movements of the ship. When attempting to determine just how these movements produce the enumerated symptoms, anyone who has had experience in testing the vestibular portion of the internal ear cannot help but be struck by the similarity of symptoms that make their appearance in individuals who are turned in a revolving-chair. Such turning may produce the identical symptom-complex produced by the same cause—movement of the body. In the one instance the movement is produced by the tossing of the ship; in the other it is produced by being turned in a chair. We know that being revolved in a chair stimulates the hair-cells in the semicircular canals by producing a movement of the lymph within the canals. At the outset, therefore, it is fair to assume that those vague symptoms known as *mal-de-mer* are produced in the same way. Furthermore, if this assumption be correct, *any* agency which induces movement of the lymph within the semicircular canals should produce the same symptoms. Clinical experience tells us that this is true; *douching* the ear with either cold or hot water produces the same train of symptoms. Chilling the outer portion of the capsule of the internal ear by douching with cold water causes a downward flow of the endolymph, whereas hot water causes the endolymph to rise. Continued douching with cold or hot water inevitably causes any normal individual to be violently "seasick." With these thoughts in mind, Ruttin conducted the following experiment upon himself: he purposely went out to sea in order to make himself seasick and when in that condition he had both of his ears douched simultaneously by means of a double irrigator devised for the purpose. The seasickness disappeared while his ears were being douched, but unfortunately immediately returned after the douching was stopped. The powerful effect of the douching had caused a continuous flow of the endolymph in one direction and for the time being made it impossible for his internal ears to detect the varied movements of the ship.

Very few individuals are exempt from seasickness: women are more susceptible than men, little children are very little affected and infants are immune. It is striking to note that artificial stimulation of the internal ear, either by turning or douching, produces a more marked effect in women than in men, very little effect in little children and no effect in infants. We have never seen vomiting result from douching an infant's ear, no matter how long the douching was continued. The violence of response to the movement of the lymph within the internal ear, whether due to the tossing of the ship or to turning and douching, is in direct proportion to the degree of activity of this organ. The vestibular mechanism of an infant is not yet fully developed and it is for this reason that the infant is immune to seasickness.

Further analogies between the movement of the ship and the result of artificial stimulation of the ear are brought out by Barany.[1] In both instances the symptoms subside with the removal of the cause—the docking of the ship in the one case and a cessation of labyrinthine stimulation in the other. Alteration of the position of the head affects the intensity of the symptoms in both instances. Nervous or high-strung individuals are more susceptible in both instances. Individuals acquire a tolerance to the movement of the ship so that they gradually lose their susceptibility to seasickness; in the same way a tolerance is induced for the tests of the internal ear by repeating them a number of times. An individual who cannot be nauseated by repeated examinations of the vestibular apparatus cannot be made seasick. This fact has been repeatedly borne out in our experience. Furthermore, if a certain plane of movement of the ship is found to affect an individual more than any other it will be found that the particular form of vestibular stimulation which imitates the same movement will also affect the individual more than a movement in another plane.

Any physician who has been violently seasick and has experienced the sense of utter annihilation, with violent nausea and vomiting, diarrhœa and perspiration, immediately recognizes that these symptoms are identical with phenomena seen in cases of *pathological* conditions of the internal ear, such as the various forms of labyrinthitis—the so-called "Meniere's disease." Here then we have clinical evidence of the similarity between seasickness and an internal ear-disturbance.

All these points of resemblance seem to point to *mal-de-mer* as being unquestionably an ear-phenomenon. This is finally and definitely proved by the observations of James and Kreidl. The former noted that deaf mutes could not be made seasick under any circumstances. With this in mind, Kreidl experimented upon animals which he rocked or revolved upon a mechanical platform until they became seasick. He afterwards operated upon these animals and severed their VIII Nerves. After they had recovered, he repeated the same experiment and was unable to make them vomit even by the most violent movements.

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[1] Seekrankheit, Handbuch der Neurologie, 1914.

Seasickness therefore is an ear-phenomenon. We now consider the mechanism that produces this phenomenon. The train of symptoms known as *mal-de-mer* is the same as that seen after prolonged and violent stimulation of the ears; the rolling and pitching movements of the ship constitute the cause of the ear-stimulation. To be sure, the ear of itself does not produce nausea or vomiting and the other symptoms; these manifestations are brought on by a disturbance of the central nervous system. The equilibratory end-organ consists of two tiny sacs, the saccule and utricle, and three minute tubes, the semicircular canals; these sacs and tubes are so designed as to take cognizance of every conceivable movement of the body. There are two of these organs, one on each side of the head, each one continually sending out a flow of tonic impulses to various parts of the central nervous system and also to the musculature of the entire body. Movement of the lymph in these sacs or tubes in one direction stimulates one group of hair-cells, producing certain phenomena; movement in a reverse direction affects different hair-cells in the end-organ and results in different phenomena. This is made possible because of different nerve-pathways connecting the different hair-cells with individual and separate central nuclei. Some of these nerve-paths have been actually traced from the ear to the various nuclei in the brain-stem and cerebellum. Ordinary movements of the body produce relatively different movements of the endolymph in the semicircular canals on both sides, but there is no confusion because the brain, through countless repetitions, has learned to interpret the significance of all these movements. Experiments made on individuals in turning-chairs, swings, or in rapidly-moving elevators, show that a continuous definite stimulation of one or more semicircular canals, produced by an uninterrupted flow of endolymph, is usually unaccompanied by any unpleasantness; disturbances occur as soon as there are reversals of the relation between the moving endolymph and the hair-cells, such as happen when there are repeated cessations in the stimulation, especially if such stops and starts are carried out in quick succession. The same is true when the endolymph is set in motion by douching the ear. If the right ear with the head back is douched with cold water, a current is created in a downward direction to the right; if the head is then quickly put 120° forward, the current of the endolymph is reversed to the left. Putting the head back again, again reverses the current. Now an individual may not be at all affected by having his ear douched with the head back 60°, provided it remain in that position as long as the stimulation lasts. If, however, the position of the head be changed a number of times, the symptoms of seasickness invariably appear. On board a vessel we unfortunately have just such a state of affairs, in which the complexity of movement is forever changing the direction of the endolymph-current in the semicircular canals, creating havoc with the nerve-centres.

The state of brain-confusion and lack of interpretation of these impulses does not last forever, and after a varying length of time, longer with some individuals and shorter with others, a readjustment occurs. The brain learns the meaning of all these stimuli, and an immunity from seasickness results. Such an immunity is not permanent and consists only of an exemption from the malady under conditions similar to those to which the individual had become accustomed; one may become immune to seasickness when aboard a large vessel sailing on the ocean, and yet when embarking on a smaller ship, on a land-locked sea, the movements he is then subjected to are quite different from those to which he had become accustomed, and seasickness may again result.

The mechanism of vomiting consists chiefly of motor impulses through the vagus and phrenic nerves. The nausea is an entirely different phenomenon, and consists of a cerebral interpretation of the unusual stimuli. Cumulative nausea may produce vomiting by a motor impulse from the cerebral cortex to the nuclei of the vagus and phrenic nerves. This is apparently the usual sequence seen in seasickness; an attack almost always begins with a vague unpleasantness which gradually develops into a nausea which increases in severity little by little until suddenly projectile vomiting occurs—as the result apparently of the cumulative nausea. At first glance it would appear that the vomiting is necessarily the result of cerebral impulses; six clinical cases in our experience, however, show that cerebral impulses are not necessary for the stimulation of the vagus and phrenic nerves and that there must be not only direct pathways between the ear and the vagus and phrenic nuclei, but also that along these pathways alone stimuli may produce projectile vomiting entirely dissociated with any cerebral phenomena. In these six cases because of lesions high up in the brain-stem, stimulation of the ears failed to produce the normal responses; prolonged stimulation produced no nystagmus, no vertigo, no past-pointing and no falling—there was no nausea and the patient in fact asserted, in each of these six cases, that he did not in any way "feel different"—and yet suddenly violent projectile *vomiting* occurred. This phenomenon makes it evident that there is a lower direct reflex pathway between the semicircular canals and the nuclei of the vagus and phrenic nerves. It is very easy to conceive the probable course of these pathways. Certain vestibular fibres are known to enter the posterior longitudinal bundle, which in turn is directly connected with the vagus nucleus; this is the probable anatomical pathway from the ear to the Xth nucleus. Other vestibular fibres after entering the medulla oblongata are known to descend to the motor cells of the anterior horns of the spinal cord; the phrenic nerve has its origin in the cells of the anterior horns of the spinal cord at the second, third and fourth cervical vertebræ. It is very easy to see, therefore, how impulses from the ear may travel directly to the phrenic nerve.

It must of course be recognized that there are many associated and predisposing factors in the production of seasickness apart from the actual movement of the endolymph within the semicircular canals. Impressions received through other special senses may play a certain part. It is a fact of common knowledge that the presence of certain odors exert a definite influence in increasing the nausea. Many individuals feel that they could have weathered a trip in fair comfort if it had not been for certain odors, such as the smell of food, particularly "greasy" food, such as pork fat, or the so-called "ship's odors." In a similar way visual impressions of the movement of the waves or of the line of the horizon, or the mere sight of other people suffering from seasickness, or looking at the vomitus from another sufferer, may bring on an attack. These olfactory and visual impulses as a rule merely constitute the "last straw" in overcoming the cerebral attempt to keep from vomiting, by increasing the nausea through the unpleasant odors or visual impressions. Cases are known, however, in which the mere sight of a ship at her moorings has brought on nausea and vomiting. Mere fear of the malady itself is at times a very potent factor; such a psychic state no doubt increases the irritability of the nervous mechanism. The essential mechanism of seasickness, however, is that movement of the endolymph within the internal ears produces nausea by means of impulses from the ears to the cerebral cortex, and produces vomiting not only as a secondary response to the nausea through cerebral impulses to the vagus and phrenic nerves, but also by direct impulses from the ears to the nerves whose hyperactivity produces the actual vomiting.

Barany has classified the various movements of the ship as occurring in certain planes. When the passenger is standing erect, any movement of the ship in the horizontal plane affects his horizontal semicircular canals, and produces no unpleasantness. Unfortunately this movement of the ship is very rarely seen. The rolling of the ship from side to side affects his vertical semicircular canals (if he be facing bow or stern) in the frontal plane; this is distinctly unpleasant, and if under these circumstances the individual will lie down in the long axis of the ship, from stem to stern, the rolling movement will then affect the horizontal canals and relieve the unpleasantness. If the ship is pitching fore and aft, and the passenger is standing upright, facing the bow or stern, his vertical semicircular canals are influenced in the sagittal plane; if he then lies down in a plane across the ship, the pitching movement will then affect his horizontal canals and thus relieve the unpleasantness. Similarly when the individual is standing, the up and down, rising and falling movement of the ship will affect him unpleasantly, and this may also be relieved by lying down. Very frequently the effect of seasickness can be greatly ameliorated by following out these simple measures; it is interesting to note that observations made of seasick people show that they intuitively assume the above positions. It is also clinically true that most people "feel better when they lie down." Testing individuals in the turning-chair corroborates these facts; turning with the head upright, stimulating the horizontal canals is not unpleasant, but turning with the head forward or backward, stimulating the vertical canals in the frontal plane, or turning with the head inclined toward the shoulder, stimulating the vertical canals in the sagittal plane, is invariably unpleasant. Naturally on board ship we are seldom dealing with movements in only one plane, and it is the combination of two or more planes of movement that renders it impossible to adapt one's self to so many varied movements.

### THE QUESTION OF PREVENTION AND TREATMENT OF SEASICKNESS

This much discussed subject is in one sense the most unique in the whole field of therapeutics, in that we are dealing with a sickness which is not a sickness in the true sense of the word. It is a normal reaction in a normal mechanism. *Seasickness* is of course a misnomer; it is no sense pathologic and is not due to "What I had for breakfast" or "Because I am in such a run-down condition." Such states to be sure may produce a nervous irritability, which would predispose the individual to seasickness; but the fundamental cause itself—varied movements of the endolymph and the inability of the brain either to interpret or disregard them—is purely physiologic. Here lies the difficulty in prevention and treatment. It is obviously impossible to prevent the movement of the endolymph. We are confronted with this physiologic phenomenon resulting from an extraordinary stimulation in a normal organ by perfectly physiologic means. With the removal of this stimulation in fact the indisposition promptly disappears. The fact remains, however, that the symptoms are most distressing and any means toward their amelioration should receive careful thought.

With the true etiology of the condition in mind, the problem presented is to find means of lessening the irritability either of the end-organs within the labyrinth, or the conducting nerve paths, or of the nuclei of the vagus and phrenic nerves. If there were, to be sure, some specific drug that could temporarily abolish the function of the internal ear or VIII Nerve, seasickness would be completely "cured"; unfortunately the pharmacopœia has no drug with such a selective action, and even if there were such a drug, the individual would probably be stone-deaf throughout the voyage. We are compelled to rely on the cautious administration of those remedies which lessen general nervous irritability, and in this way also influence the nervous mechanism in question. The bromides are the most valuable of all remedies for the prevention and amelioration of seasickness. The strontium, ammonium, sodium and potassium salts, singly or in combination, may be employed; strontium salt is preferable as it is the least irritating to the stomach. It should be administered in moderate doses, such as 15 grains three times daily, beginning one week prior to the sailing and continued throughout the entire voyage. Other nerve sedatives, especially the derivations of opium, are also of distinct value.

In a general way it may be said that all measures directed toward the elimination of nervousness, fear and conditions conducive to lowering the vitality generally, would in a measure tend to prevent seasickness. The gastro-intestinal tract should be put in the best possible condition. A brisk cathartic on the day before sailing and a very light assimilable diet prior to starting on the voyage will be found helpful. Various digestants, such as pepsin and peptenzyme, have appeared to have a beneficial influence in certain cases, but it must be recognized that they are useful only in so far as they aid in putting the gastro-intestinal tract in as normal a condition as possible.

Swinging beds or chairs have been suggested and have been tried out, but with apparently no satisfactory result. If they were mechanically feasible, such contrivances would rob the sea of all its terrors; but when we remember the complexity of movement to which a ship is subjected, we can readily see how any swinging chair or bed to eliminate them all is an utter impossibility.

The only final and really effective means of preventing seasickness is to get the individual accustomed to the extraordinary labyrinthine stimulation. The sailor and seasoned traveler accomplish this by the many repetitions to which they subject themselves in their travels. We know, however, that it is not necessary to go out to sea in order to obtain the phenomena of labyrinthine stimulation. This can be easily accomplished in the turning-chair and with this advantage, that the amount and violence of such stimulation is under the perfect control of the doctor. An actual attack of seasickness need never be brought on; the individual can either be rotated or rocked, or have the ears douched up to the limit of toleration and the stimulation may be discontinued with the first sign of discomfort. Although such a "treatment" is more of an academic than practical interest, it is perfectly possible to accustom an individual to increasing "doses" of ear-stimulation, and in this way to cause the individual to develop a gradually increasing tolerance. Such experimental stimulation should enable an individual to go aboard a ship for the first time practically a seasoned sailor. This theory has been borne out in our clinical experience in that repeated stimulation of the ears in the same individual has invariably shown an increasing tolerance.

Before an individual goes on a voyage it is very simple by means of the turning and douching tests to determine whether or not he will be subject to seasickness; if repeated turnings with the head in different positions fails to produce unpleasant sensations of vertigo or nausea, the individual may be assured that he has nothing to fear from the movement of the waves. If, on the other hand, the turning tests prove disagreeable, the individual is very apt to become seasick during the proposed voyage. In other words, it is a very simple matter to determine the degree of function and stability of the vestibular apparatus in any given individual. If the patient shows a high degree of irritability in the responses to the tests of the turning-chair, he is then forewarned and may then be forearmed by appropriate medication before starting on the voyage.

The treatment of an actual attack of *mal-de-mer* resolves itself into the symptomatic treatment of the phenomena that present themselves at the time. The vomiting and retching are not only extremely distressing but may even result fatally from hemorrhage, especially in cases in which there is present a pathologic condition of the stomach. Although lavage is distinctly helpful, it is well to remember that a stomach tube is not very safe in cases of violent seasickness, as the slightest abrasion of the gastric mucosa may be attended with serious consequences. The same result can be accomplished by the administration of copious draughts of some alkaline solution, such as bicarbonate of soda or of a solution of salt and water. This washing of the stomach by drinking the sodium bicarbonate or salt solution helps to dissolve the ropy mucus within the stomach and also tends to relieve the retching. If the retching is very violent and shows no tendency to abate, by far the best remedy is a hypodermic injection of morphine, gr. $1/6$, to be repeated if necessary.

The character of the "sea" prevailing at the time should be studied and the patient placed so that the horizontal semicircular canals are the ones most affected. The recumbent posture in the open air should be advised if possible.

In treating the collapse which is usually felt after an attack, strychnine, gr. $1/30$, or atropin, gr. $1/120$, one or both, administered preferably by hypodermic injection, are at times helpful. As a depressor of sensory nerves all over the body and particularly in the intestinal tract, atropin helps to eliminate an important secondary source of distress. In addition to this the stimulating action of atropin on the medullary centres is a great aid in regulating the circulatory disturbances incidental to seasickness. Alcohol produces a temporary cerebral exhilaration so that for the time being the individual "feels better"; but it is well to bear in mind that alcohol unquestionably increases the irritability of the nervous system, and this is just the very thing we wish to avoid.

## CHAPTER V

## THE EAR IN SYPHILIS

It has been known for years that the syphilitic toxin shows a marked affinity for the VIII Nerve; actual impairment of hearing, or even deafness, has been observed in many cases very early in the disease. It is a common experience among otologists to find that a large percentage of syphilitics in the various stages of the disease, show an impairment of the labyrinth and VIII Nerve. In determining the degree of function of the labyrinth or VIII Nerve, however, the otologist has been relying upon the tuning-fork tests and has based his conclusions upon the following findings:

(1) Bone-conduction less than normal. The shank of a vibrating tuning-fork is held against the head; if the individual ceases to hear the sound before the tuning-fork has ceased to vibrate, the bone-conduction is less than normal.

(2) Air-conduction greater than bone-conduction, in the presence of impaired hearing. This suggests a nerve involvement because if the impaired hearing were due to a *middle* ear lesion, the air-conduction would be less than the bone-conduction.

(3) An impairment of hearing for the high tones is generally regarded as indicating an impairment of the general ear or VIII Nerve.

These tests of the auditory function, to be sure, should not be underestimated; but as regards precision in diagnosis, which is so essential in this matter of syphilis, they have left much to be desired. The diminution of bone-conduction is of itself unreliable, not only because it depends upon the statement of the patient as to his subjective sensations, but also because even in the presence of cochlear degeneration, the bone-conduction may not be diminished because of an associated obstructive lesion of the middle ear. It is perhaps because of this indefiniteness that the use of ear-examinations in syphilis has not come into general use.

In the new ear-tests, however, we are dealing with a very different matter; these tests are not only much more delicate, but constitute an *objective* measurement of the degree of function of the VIII Nerve. We are not dependent on mere subjective impressions of the patient. It is a very different matter on the one hand to say, "The patient's hearing is impaired, yet the air-conduction is less than the bone-conduction and the bone-conduction is less than normal," and on the other hand to state, "Turning to the right produced a horizontal nystagmus to the left of *11 seconds* of poor amplitude." Here we express the degree of function of the VIII Nerve with mathematical precision, and by actually observing the movement of the eyes.

The data obtained by an examination of the labyrinth are of value to the syphilologist in the following ways: (1) As an aid in the early diagnosis of syphilis; (2) in the early diagnosis of syphilis of the nervous system; (3) in the early recognition of neural recurrences; (4) in estimating therapeutic activity and efficiency; (5) in helping to determine whether a case of syphilis is cured.

(1) In the early diagnosis of syphilis the method par excellence is of course the demonstration of the spirochæta pallida in the serum obtained from the suspected initial lesion. This examination requires only the ordinary microscope with its oil immersion lens, fitted with a dark field condenser, and with a brilliant source of illumination. The demonstration of the organism within the first few days after the appearance of the suspected lesion constitutes the ideal early diagnosis of the disease. When, however, for one reason or another, as so frequently happens, such a demonstration of the micro-organism is not made, the clinician must rely for his diagnosis on the early recognition of constitutional involvement, either by the Wassermann test, the appearance of the well-recognized clinical manifestations of secondary syphilis, or by observing deviations from the normal responses to stimulation of the ear. The ear examination can be useful in the following way: If a patient presents a suspicious initial lesion and the ear-tests on the eighth or tenth day show a nystagmus, after turning, of

18 seconds instead of the normal 26 seconds, syphilis may be suspected. If after three or four days more, the after-turning nystagmus lasts only 14 seconds and a few days later only 12 or 10 seconds, the diagnosis of syphilis is strongly suggested. To be sure diseases other than syphilis may have previously caused an VIII Nerve impairment, and naturally all diseases, such as mumps, scarlet fever, diphtheria, nephritis, diabetes and gastro-intestinal intoxication must be considered. In all of these affections, however, the impairment has been produced at some previous time and the resulting impairment remains *constant* during the period of the repeated tests. A progressive impairment from day to day, on the other hand, gives evidence of a strong, active toxin. This strongly suggests syphilis.

The Wassermann reaction was justly hailed as an extraordinary aid to the clinician, and, when properly performed, it is theoretically a specific diagnostic method. Unfortunately, the Wassermann reaction is not absolute. Clinical experience teaches us that the Wassermann reaction, as performed by various laboratories, cannot be relied upon in many cases to make the diagnosis of syphilis. It is common experience to find cases that are clinically undoubtedly syphilitic, and which also improve under antisyphilitic treatment which are repeatedly negative to the Wassermann test. The Wassermann test has also been reported positive in many cases which have subsequently been proven to be non-syphilitic. It is therefore evident that in many cases we must rely upon other means of diagnosis, and in many such cases the tests of the VIII Nerve may lead to valuable conclusions as to the presence of a constitutional infection, especially so if there is found a progressive impairment in the responses obtained by repeating these tests at intervals of a few days.

(2) Although it will require years of investigation to determine the exact status of the usefulness of these tests in the early diagnosis of syphilis, we already know that in detecting a beginning involvement of the central nervous system, these tests are of unquestionable value. It is generally stated that the nervous system is attacked in five per cent. of those affected. We venture

to suggest, in the light of our experience to date, that probably this percentage is much higher, and that many cases are not recognized because there is no *obvious* involvement, as in locomotor ataxia, facial paralysis, or general paresis. The analysis of the function of the VIII Nerve and of the intracranial pathways from the VIII Nerve within the brain-stem, cerebellum, and cerebrum, may, in certain cases, demonstrate a beginning involvement of the nervous system several years before it could be detected by any other method.

The importance of the early recognition of cerebro-spinal syphilis cannot be too strongly emphasized, as only by the early recognition and appropriate treatment will the late and incurable manifestations of syphilis of the nervous system be avoided. There is both experimental and clinical evidence to show that certain types of spirochæta pallida have a special affinity for nerve tissue and infections with this type of spirochæta pallida are not so apt to produce the characteristic cutaneous lesions of secondary, and particularly tertiary, syphilis. Spirochæta pallidæ recovered from the brain and cord of ataxic and paretic patients have been inoculated into monkeys and have produced in them lesions of the nervous system, thus showing a direct affinity of this type of spirochæta pallida for nerve tissue. Clinically, we note frequently a negative history of chancre and secondary eruption in patients suffering with paresis and locomotor ataxia—diseases of unquestioned syphilitic etiology. Many such patients are married, have apparently healthy wives and children and vehemently deny the possibility of syphilitic infection. Furthermore, it is unusual to encounter upon such patients the typical scars of healed tertiary syphilides. Vice versa, patients suffering with recurrent cutaneous tertiary syphilides rarely become victims of locomotor ataxia or paresis.

The diagnosis of cerebrospinal syphilis has depended either upon the Wassermann test of the spinal fluid or the appearance of clinical evidence of involvement of the cerebrospinal system. A Wassermann test of the spinal fluid is easily made after the fluid is obtained, and although this is usually secured without difficulty by spinal puncture, it is advisable that the patient go to bed for at least twenty-four hours, and frequently there occur disagreeable sequelæ, including severe headache, nausea, vomiting and pain, all of which considerably influence the patient against subsequent spinal tappings. Involvement of the spinal nerves, manifested by alterations in sensation and in motion, occurs very late in the course of the disease, and is therefore worthless in the early recognition of syphilis of the nervous system. The same may be said of the paralyses which occur in the distribution of the cranial nerves, such as the oculo-motor, the abducens and the facial. Changes in the optic nerve also occur late in the disease, as a rule, and therefore are of little value in the early diagnosis of this disease. On the contrary, the VIII Nerve, because of its extensive ramifications and its apparent greater susceptibility to toxæmia, is the most likely of all the parts of the nervous system to be affected, at least to some extent, by this infection. Further, such changes are easily detected, and at times definitely located, by a careful analysis of the responses obtained by artificial stimulation of the vestibular portion of the VIII Nerve. There are no tests of any of the other cranial nerves which compare in delicacy and accuracy with the new tests of the VIII Nerve.

(3) The determination of neural recurrences presupposes the previous syphilitic involvement of the nervous system and its relief by suitable treatment. After the clinical signs of syphilis have disappeared, the patient enters what is called the latent stage of syphilis, and the physician is compelled to rely upon the Wassermann reaction and periodic clinical examinations as a guide to further treatment. Clinical experience teaches that when, as a result of treatment, all evidence of syphilis has disappeared and the Wassermann reaction has become negative, the first evidence of recurrence or relapse into the active stage is detected by a positive blood-serum Wassermann test. This positive reaction, if disregarded, generally increases in intensity and ultimately there occurs a clinical relapse which is frequently of a serious nature. The same condition unquestionably occurs in neural recurrences, and would probably be manifested in the same manner if a series

of Wassermann tests of the spinal fluid could be obtained as easily as a series of blood-serum reactions. If tests of the VIII Nerve prove, in the future, as valuable an index of central nervous involvement as they give promise to be, we will have in them an excellent guide as to the condition of the nervous system, of much simpler application than Wassermann tests of the spinal fluid. A negative blood-serum Wassermann cannot be relied upon to exclude involvement of the central nervous system, and many cases of well-defined lesions of the central nervous system give a positive Wassermann reaction of the spinal fluid and a negative blood-serum reaction. It therefore follows that in latent syphilis the condition of the patient should be determined by periodic Wassermann reactions of the blood-serum, by repeated tests of the VIII Nerve, and by Wassermann examinations of the cerebrospinal fluid, if permitted.

(4) In estimating the efficiency of treatment in cases of cerebrospinal syphilis, the ear-tests serve a definite purpose. In the exact measurement of the degree of function of the ear-mechanism, we have an index of the progress and extent of the specific involvement of the rest of the central nervous system. A progressive impairment in the responses to ear-stimulation indicates a change for the worse; on the other hand, a progressive improvement in the responses to the ear-tests suggests a change for the better.

(5) Before pronouncing an individual cured of syphilis, one cannot be too careful in investigating the condition of the central nervous system. The syphilologist is constantly called upon to state whether a case is cured or not. Many factors must be considered—the time at which the diagnosis was made, the promptness with which treatment was instituted, the type of treatment, repeated clinical examinations of the patient, and the results of the Wassermann tests of the blood and spinal fluid. Here again the ear-tests are of value, by providing additional information as to the condition of the central nervous system.

## CHAPTER VI

# THE EAR AND THE NEUROLOGIST

The intimate relation of the ear to the central nervous system makes the study of neuro-otology of especial interest to the neurologist. The value of an eye-examination in neurologic cases is now universally recognized. The study of the eye-grounds, the field of vision, pupillary reaction to light and accommodation and the degree of function of the eye-muscles, furnishes valuable information to the neurologist. Based on the opinion of the neurologists and ophthalmologists most acquainted with these ear-tests, it would seem safe to assert that of the two methods of approach, very much more definite information can, as a rule, be had from the examination of the vestibular apparatus than from an eye-examination. The value of ear-examination in neurologic cases is recognized in Vienna to the extent that no examination of a neurologic case is considered complete without a report from the otologist as to the condition of the vestibular apparatus.

The neurologist is not only concerned with the general problems of equilibrium which have already been presented, but, in addition, he has, in these ear-examinations, a very definite help in many perplexing diagnoses. The ear-tests are of particular value in making a differential diagnosis between labyrinth and intracranial lesions and in furnishing additional data in intracranial localization.

It is well known that nystagmus and vertigo, with loss of equilibration, associated perhaps with nausea and vomiting, may be produced either by a disturbance of the internal ear or by an intracranial lesion. In many instances the symptoms of internal ear disturbance and of a cerebellar lesion are identical. It is in such a differentiation that the ear-tests are often invaluable. It not rarely happens that a careful neurologic study indicates a lesion of the cerebellum, whereas the ear-examination, by giving additional data to the neurologist, demonstrates conclusively that he is dealing with a lesion of the labyrinth. Cases of this type are discussed on pages 19, 308, 329, 336 and 423.

A differential diagnosis between peripheral and central lesions by means of the ear-tests depends on certain general principles. A peripheral lesion, of the labyrinth or VIII Nerve, is suggested by the following:

(1) An impairment of the function of both the cochlear and kinetic-static labyrinth. If, for example, the hearing tests show cochlear deafness and the tests of the semicircular canals show that their function is also impaired, it immediately becomes probable that we are dealing with an end-organ lesion.

(2) The history or presence of tinnitus; the absence of tinnitus does not necessarily indicate that the end-organ is not involved, but its presence is very suggestive of labyrinth involvement.

(3) Proportionate impairment of the responses from the horizontal canal and of the responses from the vertical canals. If, for example, the tests show that the horizontal canal retains only one-half of its normal function and that the vertical canals similarly retain only one-half of their normal function, a lesion of the end-organ itself is suggested.

(4) Proportionate impairment of both nystagmus and vertigo. If the horizontal canal produces one-third of the normal nystagmus and one-third of the normal vertigo, it is suggested that the lesion is in the horizontal canal itself or in the fibres from the canal within the VIII Nerve; if, in addition, the vertical canals produce one-third of the normal nystagmus and one-third of the normal vertigo, an end-organ lesion is strongly suggested.

In a word, it is the "proportionate impairment" of responses that speaks for a peripheral lesion.

A *central* lesion is suggested by the following:

(1) A normal cochlea, but impaired or non-responsive semicircular canals.

(2) Normal responses from the horizontal canal, but absent responses from the vertical canals.

(3) Normal responses from the vertical canals, but impaired responses from the horizontal canal.

(4) Normal vertigo, but impaired nystagmus from the horizontal canal.

(5) Normal nystagmus, but impaired vertigo from the horizontal canal.

(6) Normal vertigo, but impaired nystagmus from the vertical canals.

(7) Normal nystagmus, but impaired vertigo from the vertical canals.

(8) Normal vertigo and normal nystagmus from any semicircular canal, but impaired *past-pointing* in any direction of any one extremity.

(9) Normal vertigo and normal nystagmus from any semicircular canal, but an impairment or absence of the normal *falling.*

(10) Spontaneous vertical nystagmus is pathognomonic of a central lesion and is indicative of involvement of the brain-stem caused either by infiltration or pressure. A lesion of the labyrinth may produce many forms of spontaneous nystagmus—horizontal, rotary, oblique or a mixed nystagmus of the various types; but an ear lesion can never produce a spontaneous vertical nystagmus, either upward or downward.

(11) If there exists a spontaneous nystagmus to the right and non-responsive semicircular canals of the right ear, an intracranial lesion is suggested. The non-responsive labyrinth, if the labyrinth itself alone were responsible, would produce a nystagmus to the left.

(12) A spontaneous nystagmus of increasing intensity or of long duration is indicative of a central lesion. A spontaneous nystagmus due to a lesion of the labyrinth shows its greatest intensity at the onset of the disease, becomes less and less marked and disappears after a few days.

(13) If a stimulation of any semicircular canal produces a "perverted" or "inverse" nystagmus, it is pathognomonic of a central lesion and is indicative of brain-stem involvement. Such phenomena as the following are frequently seen: Douching the

right ear with cold water with the head back, stimulating the right horizontal canal, should produce a pure horizontal nystagmus to the left. If on such stimulation there occurs a vertical nystagmus upward or downward, a rotary, oblique or mixed nystagmus, it may be spoken of as "perverted." If instead of a horizontal nystagmus to the left there is produced a pure horizontal nystagmus to the right, it may be termed an "inverse" nystagmus. Neither a perverted nor an inverse nystagmus can possibly be produced by a lesion of the labyrinth or VIII Nerve; a peripheral lesion produces a poor nystagmus or no nystagmus at all, but an absolutely false response of necessity demonstrates a central lesion.

(14) If ear-stimulation produces a conjugate deviation of the eyes instead of a nystagmus, it is pathognomonic of a central lesion.

The above outline indicates how additional data may be furnished to the neurologist by the ear-tests in determining whether he is dealing with a lesion of the internal ear or of the brain-stem or cerebellum. In the broader field of intracranial localization, examination of the ear and of the vestibular apparatus is also of distinct value. The particular feature of the ear-examination is that the aurist sends in a *stimulus* to the brain-centres, and then notes the *responses* of different parts of the body to this stimulus. For example, by stimulation of the ear there results a nystagmus in a given direction, a pointing of the extremities to the right or to the left as the case may be and a falling to the right, to the left, forward or backward as the case may be. Now if the ear and these nerve paths from the ear are intact, all the normal responses will appear; if there is a failure of all or any of the responses it is positive evidence of an interruption along that particular path or paths that fail to bring about these responses.

In order to utilize the knowledge obtained from these tests it is essential to have in mind the various pathways constituting the vestibular apparatus. These pathways are presented in detail in Chapters XIV, XV, and XVI. It may be stated at this point in brief that the pathways from the horizontal semicircular canal are different after entering the brain-stem from those from the vertical canals; furthermore that each set of tracts divides into two separate pathways; one pathway, the vestibulo-ocular tract, is responsible for the eye movement, and the other pathway, the vestibulo-cerebello-cerebral tract, conveys the impulses from the ear to the cerebral cortex, producing vertigo. If the horizontal canal fails to produce both nystagmus and vertigo the lesion indicated is at a point before the division of the horizontal canal fibres into their two separate pathways. Further, if the horizontal canal produces normal vertigo but no nystagmus the lesion indicated is of the vestibulo-ocular tract at a point beyond the point of division into the two paths. If the horizontal canal produces normal nystagmus but no vertigo the lesion indicated is at a point along the vestibulo-cerebello-cerebral path beyond the point of division into the two pathways. Similarly if the vertical canals produce normal vertigo but no nystagmus the lesion indicated is in the vestibulo-ocular tract at a point beyond the division into the two paths. If the vertical canals produce normal nystagmus but no vertigo, the lesion indicated is at a point along the vestibulo-cerebello-cerebral path beyond the point of division into the two pathways. A more detailed analysis of this kind is given in Chapter XXIII. The ear-tests have proved themselves surprisingly helpful in locating lesions in the cerebello-pontile angle, medulla oblongata, pons, cerebellar peduncles, cerebellum and various portions of the cerebrum, including the parietal lobe, the temporal lobe and the occipital lobe.

In order to obtain reliable data from an ear-examination, it is essential that the technic of examination should be accurate and painstaking; since it is primarily an ear-examination, the otologist is peculiarly fitted to carry out such examinations. Although one purpose of this book is to furnish a practical guide for the otologist in undertaking the examination of patients, it is also offered to the neurologist so that he, on his part, may become familiar with the ear aspects of the work. in order to realize the significance of the reactions as reported to him. The ear-examination is obviously not for the purpose of making a neurologic diagnosis; it merely gives additional information by a series of refined experiments, to the other methods at the command of the neurologist. To be sure, there are many cases in which the neurologist, without the aid of the ear, eye, blood, or other examinations, finds no difficulty in arriving at a satisfactory diagnosis. In these cases, however, it is of course useful to have the additional evidence from the ear-tests corroborating his neurologic and other findings. In addition, it not infrequently happens, in obscure cases, or in cases in which the neurologic data are meagre, that the ear-tests may be the only means of furnishing information upon which a diagnosis can be made. For example, an apparently strong, vigorous man, complaining only of headache, showed on examination, that both internal ears were normal, and also that both VIII Nerves were normal, and yet the vertical semicircular canals of both ears, when stimulated, failed to produce any responses whatever. The horizontal canals produced normal nystagmus, but no vertigo. As the labyrinths and VIII Nerves in this case were unquestionably normal, the non-appearance of the normal responses to stimulation could be accounted for only by an interference with the fibres from the labyrinth within the brain-stem. This particular phenomenon-complex indicates pressure within the IV Ventricle. This conclusion was recorded with considerable misgiving because a lesion within the IV Ventricle appeared ridiculous, in view of the man's apparent health. That night the patient was rushed to the hospital unconscious. The next day he regained consciousness, but complained of agonizing headache. Examination by a number of internists and neurologists failed to give any clue of an organic lesion anywhere, and the diagnosis of hysteria was made. Autopsy three days later showed abscess in the IV Ventricle. It is in such cases as this that the ear-examination is of the utmost importance, as it gives data absolutely unobtainable by the usual neurologic tests.

# CHAPTER VII

## THE EAR AND THE SURGEON

The diagnosis of the precise location of lesions within the cranium is probably the most difficult task with which the surgeon is confronted. Fully realizing all the difficulties in these cases, he utilizes every modern aid in diagnosis—the laboratory, the X-ray, or any other instruments of precision that may be available. For many years the ophthalmologist has been of great help to the surgeon in his intracranial cases, and no one would think of operating on such a case without first having the ophthalmologist's report as to his findings. The previous chapter has shown how the ear-tests may furnish data of value in the accurate localization of lesions which involve any of the vestibular pathways, as well as in differentiating between labyrinth and intracranial lesions. A point worthy of special emphasis is that, with the aid of these tests, he is frequently enabled to determine whether a lesion is operable or inoperable. Many lesions of the medulla oblongata, pons, or cerebellar peduncles, which are inoperable by the very nature of their location, will show very pronounced cerebellar symptoms, and not infrequently the cerebellum is explored, in such cases, in the hope of finding a tumor near the cortex, and removing it. For such a differentiation, in many cases, no method can equal the accuracy of the Barany tests. If, after turning and douching, there appears a normal past-pointing of both extremities in both directions, it may safely be assumed that the cerebellum itself is intact. This cannot be regarded as absolutely final, but is much more definite than any other known method for determining the integrity of the cerebellum. It is perfectly conceivable that a case showing almost normal past-pointing may apparently reveal a lesion when a cerebellar decompression is done. We have seen two such cases. Here, however, it should be explained that in both these instances the lesion proved to be a cyst which grew from the brain-stem and extended backwards between the cerebellar hemispheres, without, however, destroying the substance of the cerebellum itself. Such cases give definite neurologic evidence of a cerebellar lesion, and at the time of operation, such a lesion is apparently discovered. There may have been present asynergy, manifested by hypermetry, adiodokokinesis and tremor, yet these symptoms or phenomena were produced by involvement of the fibres *on their way to* the cerebellum, or by pressure upon the cerebellum itself. If, then, the ear-tests demonstrate normal past-pointing, it is strong evidence that the cerebellum itself is not involved.

Tumors in the cerebello-pontile angle either originate from one of the cranial nerves in the angle, usually the VIII Nerve, or have invaded the angle secondarily from the cerebellum or brain-stem. These tumors invading the angle from other adjacent structures also usually involve the VIII Nerve. Obviously then a careful study of the various portions of the VIII Nerve gives direct insight as to conditions in the cerebello-pontile angle. In addition, in our experience the ear-tests have usually demonstrated the two following phenomena, in cases of cerebello-pontile angle growth:

(1) An absence of all responses from the vertical canals of the ear *opposite* the side of the lesion. Given a tumor in the right cerebello-pontile angle, the usual findings are as follows: The right ear gives no responses—the cochlea shows absence of all function and the horizontal and vertical canals fail to produce any nystagmus, vertigo, past-pointing or falling. The left ear shows unimpaired hearing and the left horizontal canal does produce nystagmus, vertigo, past-pointing and falling. The left vertical canals, however, fail to produce any responses, because the vertical canals fibres are most probably impaired because of pressure upon the pons by the tumor in the angle.

(2) "Crossed past-pointing." This phenomenon consists of persistent past-pointing of both upper extremities either outward or inward, regardless of the type of ear-stimulation employed.

If ear-stimulation produces normal nystagmus, vertigo, past-pointing and falling, the Barany tests are of unquestioned value in eliminating lesions in the posterior fossa and brain-stem. If they rendered no other service than this it would be sufficient to hail them as a distinct contribution in the diagnosis of intracranial lesions. In addition these tests may prove helpful to the surgeon in preventing unnecessary operations. One case, given in detail in Chapter XXIII, page 308, case number 2, is of interest in this connection. A woman appeared to have a tumor of the right cerebellar hemisphere. This diagnosis was confirmed by neurologic consultants and the X-ray report stated that it was a cyst in the right cerebellar hemisphere. The ear-tests suggested that the cerebellum was uninvolved; operation was delayed on this account and eventually was found to be unnecessary, as the patient steadily improved and was discharged from the hospital apparently well and for years has had no recurrence of her symptoms. Her cerebellar symptoms evidently were due to a disturbance in the internal ear.

No operation upon the brain should be undertaken without giving the patient the benefit of an ear-examination.

## CHAPTER VIII

## THE EAR AND THE OPHTHALMOLOGIST

The intimate relation between the ear and the eye can be best appreciated when we realize that the ocular mechanism constantly depends upon stimuli from the ear for precision of movement. Steadiness of fixation is dependent to a large extent on normally functionating ears. Tonic impulses from the right ear continually tend to draw both eyes to the left. This is definitely proven in that a sudden loss of function of the right internal ear invariably results in a deviation of the eyes to the right, because the tonic impulses tending to draw the eyes to the left is impaired, and there is a resulting *nystagmus* to the left. Similarly sudden loss of function of the left internal ear invariably results in a deviation of the eyes to the left, because the tonic impulse tending to draw the eyes to the right is impaired, and there is a resulting *nystagmus* to the right. This may be further demonstrated experimentally by the use of electricity. In applying the galvanic current to the right ear, by the use of the anode, which depresses its function, there results a drawing of both eyes to the right with a resulting nystagmus to the left. The kathode, stimulating the right ear causes a drawing of both eyes to the left with a resulting nystagmus to the right. These diametrically opposite phenomena can be produced merely by reversal of the current. It is thus shown that ocular equilibrium in the same sense as bodily equilibrium is normally dependent on properly functionating ears. Barthels, in fact, has demonstrated that rabbits probably have no voluntary control of the eyes at all and that their ocular movements depend entirely upon stimuli from the internal ears; section of the VIII Nerves produces a complete loss of eye-movements.

It is also of interest to the ophthalmologist that artificial stimulation of the ear can produce "to order" a nystagmus of any type, in any direction. There is an absolute and definite relation between the canal stimulated and the type of nystagmus produced. The laws governing this relation are simple and invariable. If we wish to produce a vertical nystagmus upward, we place the patient's head over toward the right shoulder, turn him to the left, and there appears a vertical nystagmus upward. If we wish a rotary nystagmus to the left, we douche the right ear with head upright with cold water, or douche the left ear with hot water, and there is produced a rotary nystagmus to the left. These examples illustrate this marvelously exact ear-to-eye mechanism and give some conception of the fixed relationship between the semicircular canals and the eye-muscles. The "fistula" test serves as another illustration. If there is caries of bone in the outer wall of the labyrinth producing a fistula into the internal ear, or for that matter, if the stapes is unduly mobile in the oval window, applying a Politzer bag into the ear and producing pressure or suction, causes the eyes to dance violently.

These tests have a practical application in the study of ocular palsies and of spontaneous nystagmus.

## Ocular Palsies

In the study of *ocular palsies,* it is important to note that the ear-stimulation is much stronger than the voluntary cerebral stimulus. If an individual is unable to look to the right with the right eye, the question arises as to the degree of involvement of the right VI Nerve. If ear-stimulation causes the eye to move outward it is evident that the VI Nerve is not completely destroyed. This makes a refinement in the determination of the degree of paresis and to that extent is of service in estimating the prognosis. In this case in which the patient is unable to look to the right with the right eye, the right abducens nerve or its nucleus, or the right external rectus muscle are naturally suspected. To utilize the ear-tests in examining this mechanism it is only necessary to recall that a stimulation of one or the other horizontal semicircular canal can produce a movement of the right eye outward. The horizontal canal is connected by nerve paths with the VI Nucleus and with that portion of the III Nucleus which controls the internal rectus muscle. Appropriate stimulation is therefore applied, such as turning the patient in a revolving-chair to the right with the head 30° forward, or douching the right ear with head back, water 68° F. or douching the left ear, with head back, 60°, with water 112° F. All these tests normally would cause both eyes to be drawn to the right. In many cases such tests will demonstrate that the patient's inability to look to the right when asked to do so was due to only a partial involvement of the VI Nerve mechanism because when a powerful ear-stimulus was applied, the right eye did actually move to the right.

Similarly, if the patient is unable to look to the left, with the left eye, those tests are applied to the ears which should normally draw the eyes to the left, such as turning to the left with the head 30° forward, or douching the right ear with the head 60° back, with water 112° F., or douching the left ear with the head back 60°, with water 68° F.

If the patient is unable to look upward, that ear-stimulation is applied which would normally cause the eyes to be drawn upward, such as turning the patient to the right with the head inclined toward the right shoulder, or turning to the left with the head inclined toward the left shoulder. These methods illustrate how the ear-tests can determine whether any given portion of the ocular muscle mechanism is or is not destroyed.

Loss of conjugate deviation in various directions should always be studied by means of the ear-tests. There is a loss of voluntary control; the patient is unable, when asked, to look in a given direction with either eye. If the ear-stimulus causes the eyes to move in this direction, it shows without question that there are normal pathways from the ear through the eye-muscle nuclei and through the nerves of the eye-muscles themselves—therefore, the lesion is supranuclear.

## Spontaneous Nystagmus

Before taking up the value of ear-examinations in cases of spontaneous nystagmus, it must be pointed out that until recently our knowledge of nystagmus as such has been vague. It was known to occur in certain eye-conditions, was considered as a symptom of certain diseases of the central nervous system and in an indefinite way it was also noted that at times nystagmus resulted from an internal ear involvement. The new ear-tests have thrown much light upon this subject.

In a given case of spontaneous nystagmus, the first problem is the determination of its type. Is it ocular or vestibular? If the oscillations of the eye-balls consist simply of a to-and-fro roll, similar to the swing of a pendulum—that is the movement or roll in one direction is just as large and just as rapid as in the other direction—then it is of an ocular type and suggests some local eye disturbance. It may be due to any condition that prevents the rays of light from pursuing an uninterrupted course through the various ocular media, such as corneal scars or anterior polar cataract. In such a condition the nystagmus is apparently the result of an attempt to obtain a clear unobstructed view for fixation—to "look around the obstacle," as it were. Ocular nystagmus may also result from an interference with the proper action of the rays of light at the *macula,* provided that the lesion occurred before central fixation was established.

In sharp contrast, if the movements of the eyes exhibit a definite rhythm—a slow movement in one direction followed by a quick recoil in the opposite direction—the nystagmus is of a "vestibular type" and is due to a disturbance of the ocular nerve-mechanism and not to a lesion in the eyes themselves. It can be produced by a lesion affecting any of the pathways between the ear and the eye-muscle nuclei, the lesion being located either within the ear or within the cranium. Such a nystagmus is not due to an attempt to accomplish fixation but is due to a disturbance within either the nuclei themselves or through impulses from the ear to the eyes which cause the eyes to *deviate* to one side. When the eyes are thus drawn to one side, impulses from the cerebrum to the eye-muscle nuclei quickly bring the eyes back in the opposite direction. The slow component of the nystagmus is caused by an irritation, impairment or destruction of the pathways from the ear to the eye-muscle nuclei, whereas the quick component of nystagmus results from the attempt of the cerebrum to correct the altered position of the eyes.

The cause of ocular nystagmus is readily determined by an eye-examination, but it is in those cases in which the lesion lies not so near the surface that the ear-tests are absolutely essential. Recently there were two babies, both showing a wild oscillatory nystagmus. Naturally it was the ophthalmologist who was called on to determine its cause; he found nothing wrong in the eyes themselves to account for the nystagmus. The ear-tests, however, showed a striking difference between the two cases. On one we reported, ''The ear-tests suggest an irritation of the vestibulo-ocular tracts, but no organic impairment; the prognosis is good.'' The child had a perfect recovery. The report of the other patient was, ''There is an organic block of the vestibulo-ocular tracts of both sides in the brain-stem; the prognosis is bad.'' This patient died. The reasons for these reports were, that the first patient, on ear-stimulation, immediately showed a change of the oscillatory nystagmus into a definite rhythmic pull. The ear-stimulation, as it were, seized both eyes and caused them to move rhythmically in the proper direction; evidently, therefore, there was no organic block of the pathways. The other patient continued to have the oscillatory nystagmus which was entirely unaffected by ear-stimulation, thus showing a definite organic block of the pathways from both ears to the eyes.

In any case of spontaneous nystagmus in which there is no obvious ocular lesion, the ear-tests provide a means of approach in determining its cause or the site of the lesion. In fact, no case of nystagmus can now be considered to have been fully studied until the ear-tests are made.

## CHAPTER IX

## THE INTERNAL EAR AND THE OTOLOGIST

VERY little need be said of the importance or uses of a study of the cochlear labyrinth to the otologist, but those otologists who have been particularly interested in a study of the kinetic-static labyrinth feel that the recent advances in neuro-otology have a practical every-day usefulness to the otologist that is not yet recognized. An examination of the internal ear is certainly incomplete if only one portion of it has been examined. Surely the kinetic-static portion of the internal ear should receive equal consideration with the cochlear. The turning and caloric tests enable us to analyze the function of the vestibular labyrinth just as the tuning-fork and other tests of hearing enable us to study the auditory. It is distinctly unfortunate that at the present time among otologists at large there are two misconceptions in regard to this study; unquestionably many aurists hesitate to undertake this work because of these misconceptions. In the first place, the impression is general that a study of the internal ear and its intracranial pathways is *neurologic* work; and in the second place, there is no question that such examinations are regarded as extremely difficult. No one disputes that an examination of the internal ear itself to determine its own integrity is a task for the otologist; Barany has made it evident, however, that in testing the internal ear we are *at the same time* testing its intracranial pathways. While it is true that information obtained as to the condition of the intracranial pathways is of neurologic value, it is nevertheless elicited by ear-tests, and for that reason is, of course, an otologic study. There is an exact analogy in the relation of the ophthalmologist to intracranial cases; the eye-examination furnishes information of value to the neurologist and yet no one considers that such a study is merely neurologic. The otologist in precisely the same way "looks into the brain" by way of the ear.

The other misconception is that this work is enormously difficult. As a matter of fact those who are familiar with this work know that it is not in any sense more difficult than *any other form of ear-examination* thoroughly done. It is the newness of the work alone that is responsible for this misconception. The entire physiology of these ear-tests, instead of being regarded as bewildering and complex, may be summed up in *four sentences,* as follows:

(1) The eyes are always drawn in the direction of the endolymph movement.

(2) The vertigo is always in a direction opposite to the endolymph movement. (*a*) Past-pointing is always in a direction opposite to the vertigo; (*b*) falling is always in a direction opposite to the vertigo.

Merely by knowing these laws the otologist is then master of the physiology of this subject. All the combinations and permutations of the responses to ear-stimulation follow these simple laws, and it is therefore not necessary for the otologist to devote years of study to this particular subject before he feels himself equipped to undertake the tests.

Just so long as the otologist looks upon this study as neurologic and as extremely complicated and abstruse, he will be depriving himself of a valuable method of *routine* examination of ear conditions, and also of large opportunities in extending his field of usefulness.

From the standpoint of the otologist, the most useful application of this study is in the routine every-day examination of ear cases. Even in arriving at a conclusion as to the condition of the *cochlea*, these tests are often invaluable. Every otologist is constantly confronted with cases of deafness in which the significance of the tuning-fork tests is obscure and inconclusive. If in these cases one can demonstrate an involvement of the kinetic-static labyrinth, which is so readily done by these tests, he has strong presumptive evidence of a similar impairment of the cochlear portion of the internal ear. Stimulation of the internal ear by turning and douching has this advantage over the accepted methods of testing the internal ear in that we have in the resulting nystagmus, past-pointing and falling, the clean-cut, quantitative, *objective* evidence of the function of the kinetic-static portion of the labyrinth and VIII Nerve. The tuning-fork and other tests of the cochlea, on the contrary, are subjective and depend entirely on the intelligence, honesty and co-operation of the patient. The ability of the examiner to see and measure responses from ear-stimulation, such as nystagmus and falling, is a different matter; it enables him to state with greater assurance, "Yes" or "No," as to the involvement of the internal ear. In cases in which the examination of the cochlea shows practically normal hearing with the single exception that there is a marked diminution of bone-conduction, the otologist has been accustomed immediately to suspect some systemic affection, such as syphilis. Here the new ear-tests are especially useful; turning or douching shows definitely whether the internal ear *is* or is *not* affected.

In routine examination of ear-cases it is, of course, not necessary to conduct all the tests. An examination lasting only two or three minutes is usually sufficient; merely by turning the patient ten times to the right and obtaining 26 seconds of after-turning nystagmus, we at once can conclude that both the static labyrinths are normal. It is best, however, to turn again to the left to make sure that the nystagmus thus obtained is also normal. Very often, however, the nystagmus will prove to be subnormal and immediately we have a hint as to the necessity for further study. We have no hesitancy in suggesting that if the otologist in routine work will make such a single test, he will be surprised to find that in many instances an entirely new light is thrown upon the condition of his patient. These tests do not produce any discomfort to the patient, provided that too many tests are not attempted at one sitting. A few words explaining the object of the tests does away with any feeling of apprehension that the patient might have.

When the otologist is confronted with surgical problems of the internal ear itself, as in inflammatory conditions of the labyrinth resulting from an extension of a suppurative process in the middle ear, it is generally recognized that the turning and caloric tests, and occasionally the fistula test, are indispensable in determining the nature and degree of involvement. These tests are the only means by which the aural surgeon can determine whether operation on the labyrinth is or is not necessary.

Vestibular symptoms produced by non-surgical affections result from lesions either of the labyrinth itself or along the intracranial nerve-pathways from the internal ears. Such a differentiation can be made only by a study of the internal ear, so that *vertigo* from whatever cause properly belongs in the domain of otology. When examining the internal ear in an attempt to determine its integrity, the turning and caloric tests furnish the necessary information in that they either do or do not produce nystagmus, vertigo, past-pointing and falling; but in order to produce these phenomena not only the internal ear but the pathways from the internal ear to the various nerve-centres in the brain must necessarily be intact. In conducting the ear-tests, therefore, we are examining not only the internal ear itself, but the vestibular apparatus within the brain. The otologist is therefore able in this way to furnish information as to the location of intracranial lesions that involve the various pathways of the vestibular apparatus. In this broader field of intracranial localization the otologist has only made a beginning. The field is large and the possibilities enormous; but it will require the combined work of many otologists throughout the world to develop such a study to the full limit of its possibilities.

Summarizing, these neuro-otologic studies are of value to the otologist in the following ways, stated in the order of their importance: (1) In the routine study of ear-cases; (2) in determining the cause of vertigo no matter what its origin may be, and (3) in intracranial localization.

# PART II

## THE STUDY OF THE INTERNAL EAR

# CHAPTER X

## THE DEVELOPMENT OF NEURO-OTOLOGY

Although it is only in the past few years that the ear has come to be recognized as the sense-organ of equilibration, yet in a general vague way certain principles in regard to equilibration have been the subject of speculation by scientists through many centuries. Even the ancients knew that the prolonged turning of an individual produced vertigo and disturbance of equilibration; however, they did not of course recognize that it was in any sense an ear-phenomenon. In fact, it was not until the past century that a knowledge of this relation became recognized. It is of interest to note that the very first work on the relation of the ear to equilibrium and nystagmus was brought forth at about the same time by two men who were working entirely independently. In 1825 Flourens made excisions of portions of the labyrinths of animals and noted that this caused movements of the eyes and definite disturbances of equilibration; Purkinje at the same time conducted experiments in turning human beings and likewise observed the resulting nystagmus and vertigo. It was Meniere who clearly recognized that attacks of vertigo have a definite clinical relationship to diseased ears. This subject was further advanced by Goltz, who in 1870 first propounded a theory of the physiology of the semicircular canals. Further advances were made by the investigations of Breuer, Mach, Crum-Brown, Bezold, Ewald, de Cyon and others. The work of these men was utilized by von Stein, of Moscow, and Högyes, a Hungarian, who made the first systematized contribution to the study of the internal ear. The studies of Högyes represent a life work of earnest effort and unbiased observation, which were not translated from the Hungarian until a few years ago, but were carefully studied in the original by Robert Barany and was one of the contributions which made possible the phenomenal work of Barany. The efforts of the very earliest investigators were entirely along scientific lines; the practical application of these investigations fell to the lot of the Vienna School of Otologists, notably Barany, Neumann, Ruttin, Alexander and Kreidl. These men have set the stamp of Vienna indelibly upon the study of the kinetic-static labyrinth. In the past ten years Robert Barany especially has done his monumental work in extending the field of this study into the realm of neurology. For this contribution he received the prize of the year from the Society of German Neurologists, 1913, which is of particular significance in that it was an ear specialist who received a neurologic prize. The awarding of the Nobel prize to Barany two years later was the first general world-wide recognition of the great clinical importance of these studies of the internal ear.

The work of Barany in this connection is so unique that it is of interest to give his own version of how he first made these discoveries. In 1905 Barany observed that douching suppurating ears with water that was either too cold or too hot, not only produced vertigo and nystagmus but that the nystagmus was of a definite type and in a definite direction. He noted further that the direction of the nystagmus produced by water that was too cold was in a diametrically opposite direction to the nystagmus produced by water that was too hot. He then douched the ears of normal individuals (with intact drum-heads) and produced the same results. It was on the basis of these observations that Barany gave to the world the caloric test.

The next advance in this field was the contribution of Ramon y Cajal who, in 1909, demonstrated histologically that the vestibular fibres from the internal ear continue into the cerebellum. Barany was impressed with the significance of this discovery and realized the possibilities of ear-study in relation to the cerebellum. It had been only a few years previous, in 1906, that Bolk had advanced the theory of cerebellar localization, based upon researches in comparative anatomy. It was but a step for Barany to link together the work of Cajal and Bolk and work out his own conception of the uses of ear-study in cerebellar localization.

In the United States, the most important contributions to this study have been made by Mills, Shambaugh, E. R. Lewis, George

Mackenzie, Friesner, Braun, J. G. Wilson and Pike. Mills, in his textbook on Diseases of the Nervous System discussed at length the affections of equilibration due to disease of the vestibular nerve and its related encephalic structures; this is one of the first, if not the first instance, in which an effort was made to group the facts relating to the symptomatology of affections of the vestibular nerve and its correlated tracts. Shambaugh, upon the basis of extensive histologic work, has made many original contributions to the subject of the minute anatomy of the internal ear, especially in showing that there are more hair-cells on one side of each crista than on the other and in demonstrating the relation of the cupula to the hair-cells of the crista. E. R. Lewis has made the most valuable contributions in this country to the "mechanics" of vestibular action and made clear the co-working of the two vertical semicircular canals. George Mackenzie has worked out to its highest efficiency the uses of the galvanic tests of the internal ear, and also has made original observations regarding the normal duration of after-turning nystagmus; Friesner and Braun, in their books, "The Labyrinth" and "Cerebellar Abscess," have written the first comprehensive works in English on these subjects; and Wilson and Pike, by their experimental work on animals, have greatly advanced the study of the relation of the ear to the central nervous system.

## CHAPTER XI

# ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS OF THE LABYRINTH AND VIII NERVE

In order to examine the internal ear and other portions of the vestibular apparatus intelligently, and to interpret the significance of the findings, it is absolutely necessary to understand the essential features of the anatomy and physiology of the parts involved. It is our definite purpose to present only those considerations of the anatomy and physiology which deal directly with the relation of the ear to the central nervous system. The otologist is naturally familiar with the structure and the functions of the internal ear; this chapter is therefore written more especially for the general practitioner, ophthalmologist, syphilologist, neurologist and surgeon, in order that they may be reminded of the fundamental principles underlying the tests of the labyrinth. Having once visualized the mechanism of the internal ear, one is then in a position not only to perform the tests but to understand and interpret the resulting phenomena.

### Anatomic Considerations

The labyrinth is a series of intercommunicating spaces situated within the petrous portion of the temporal bone, and contains a membranous sac known as the membranous labyrinth. The bony labyrinth consists of a central chamber called the vestibule from which there extend anteriorly a coiled tube known as the cochlea and posteriorly three tubes known as the semicircular canals (Fig. 3).

The cochlear tube is arranged in a spiral of two and a half turns. The three semicircular canals are placed at right angles to each other in three different planes of space, and are known as the "horizontal" or external canal, the superior (vertical) canal and the posterior (vertical) canal. The superior canal lies in a plane halfway between the frontal and the sagittal; the outer-

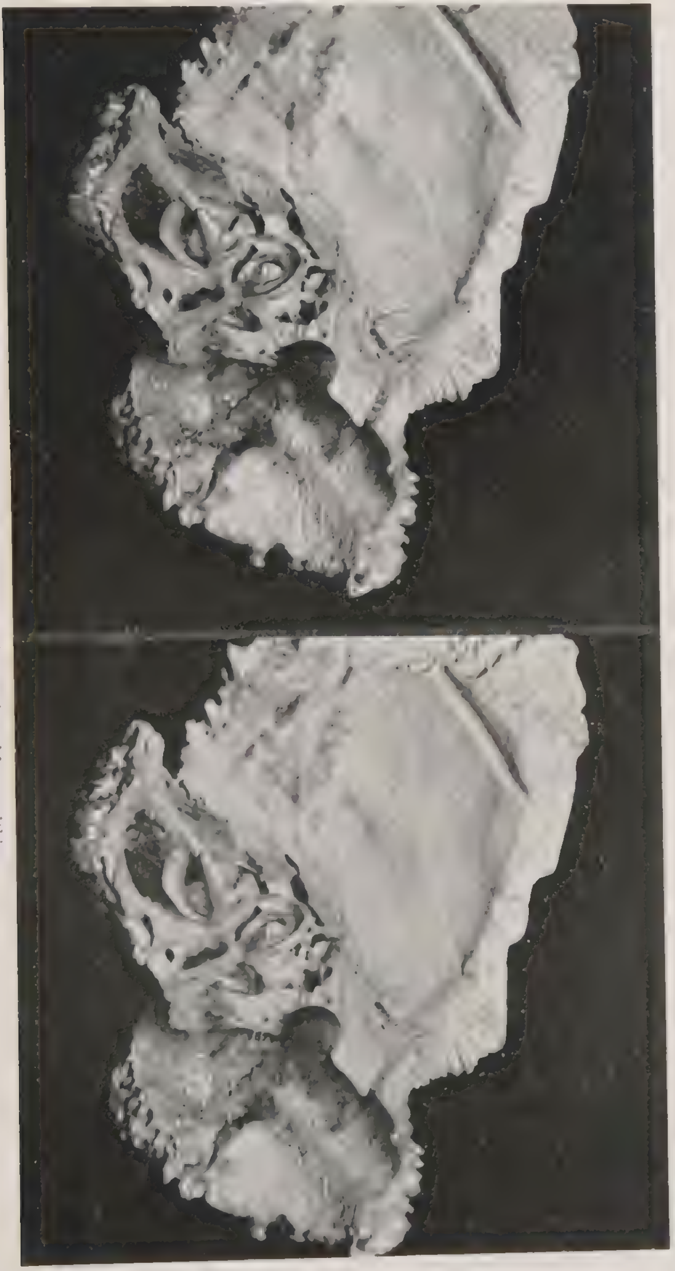


Fig. 3.—Right internal ear, viewed from within.

most portion is placed anteriorly and the plane continues inward and backward. The posterior canal also lies in a plane halfway between the frontal and the sagittal; the outermost portion, however, is placed posteriorly and the plane continues forward and inward. The superior canal of the right ear and the posterior canal of the left ear are situated in parallel planes; similarly the superior canal of the left ear and the posterior canal of the right ear occupy parallel planes. When the head is inclined *30 degrees forward* the horizontal canal then lies in a

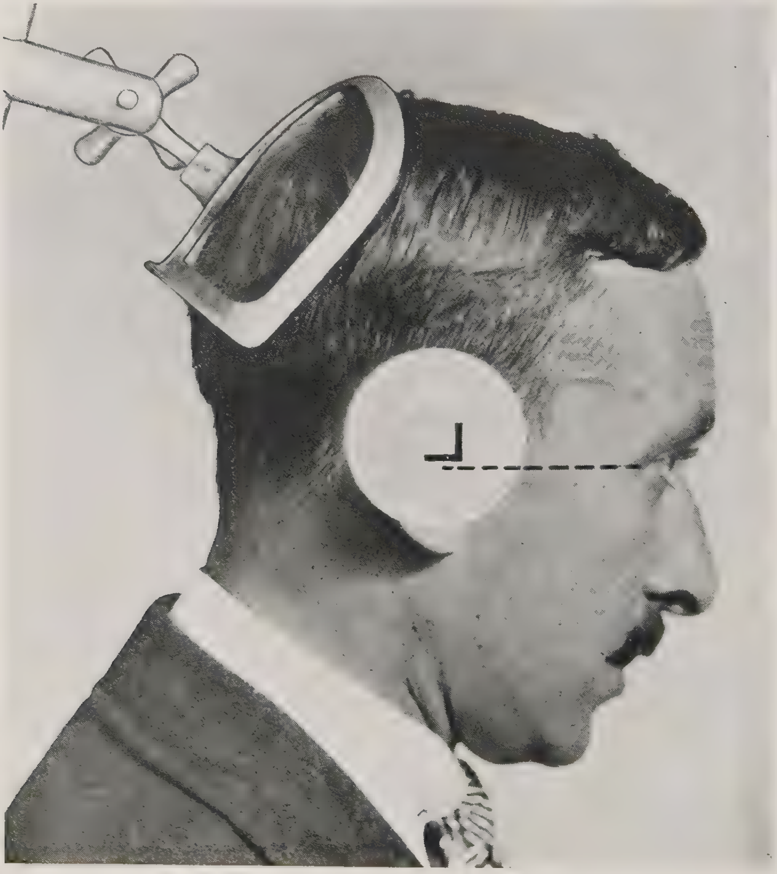


Fig. 4.—Head inclined 30° forward. Dotted line between the external auditory canal and the external canthus is parallel to the floor; this places horizontal canal in a horizontal plane and vertical canals in a vertical plane.

plane exactly parallel with the floor, and the vertical canals, the superior and posterior, are at exactly right angles to the floor.

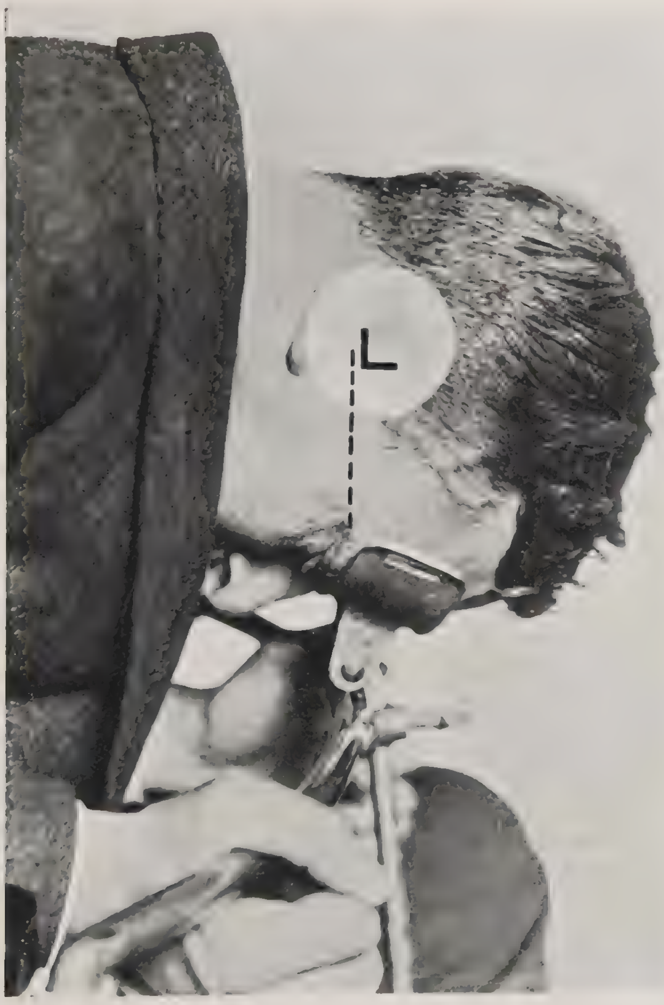


Fig. 5.—Head inclined 120° forward. Dotted line between the external auditory meatus and the external canthus is at right angles to the floor; this places horizontal canal in a vertical plane and the vertical canals in a horizontal plane.

This position of the head is important to bear in mind, as it must be used routinely in testing the semicircular canals (Fig. 4, Fig. 5, and Fig. 6).

Each semicircular canal has at one end a bulbous swelling known as the *ampulla*. The other end of the horizontal canal leads directly into the vestibule, whereas the smooth non-ampullated ends of the superior and posterior canals unite in a common tube which in turn opens into the vestibule.

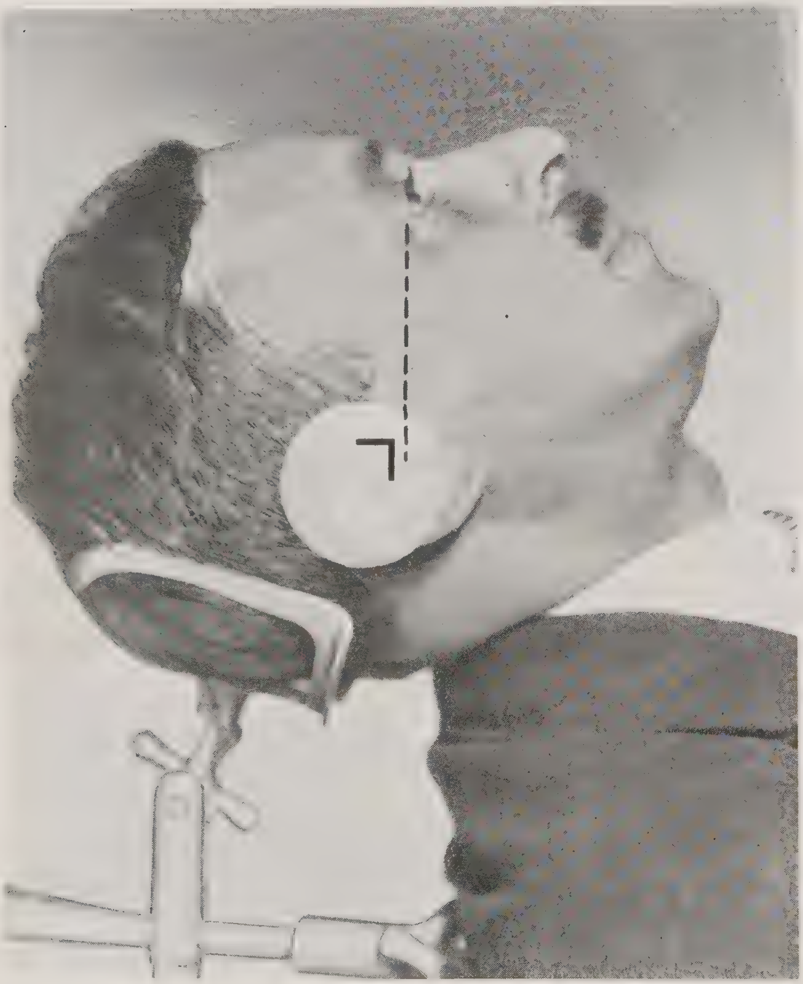


Fig. 6.—Head inclined 60° backward. Dotted line between the external auditory meatus and the external canthus is at right angles to the floor; this places the horizontal canal in a vertical plane and the vertical canals in a horizontal plane.

The membranous labyrinth is the true labyrinth and is embryologically the primary formation; the bony capsule is a later development and conforms itself to the general shape of the original membranous labyrinth. Primarily in the lower forms of life as as well as in the embryo, the internal ear consists of a small sac, which eventually in the higher forms of life differentiates into an anterior cochlear portion and a posterior vestibular portion. In each animal this ear-sac develops that portion which is most essential to its environment—the fish develops semicircular canals but has no cochlea; the bird has a very highly developed semicircular canal system and also has a cochlea. It is in mammals and man that the cochlea, in addition to the other portions of the internal ear, attains its highest development.

The membranous labyrinth occupies approximately one-third of the lumen of the bony labyrinth and is attached to the bony walls by fibrous bands at the point of exit of the nerve-fibres. It is a counterpart of the bony labyrinth in all its parts except that portion lying within the vestibule; this portion (corresponding to the vestibule) is subdivided into two sacs known as the *saccule* and the *utricle.* The membranous labyrinth is a closed sac and contains a fluid of high specific gravity, the *endolymph,* which has no means of escape. This entire structure floats in a fluid of lower specific gravity known as the *perilymph,* which fills the bony labyrinth; in contrast with the endolymph, this perilymph does have a means of escape, having a direct communication with the rest of the cerebro-spinal fluid through a small tube, which opens into a subarachnoid space.

The actual sense-organs of the internal ear are contained within the membranous labyrinth. The labyrinthine end-organ is essentially a hair-cell adapted to receive stimuli from wave-impulses of the endolymph impinging upon the hairs. Within the cochlea these hair-cells are grouped into an auditory apparatus known as the *Organ of Corti;* 3000 to 6000 units of Corti's organ extend throughout the entire cochlear tube. The second type of end-organ, known as the *macula,* is found in the saccule and the utricle, one in each. The third type of the end-organ is found in the ampulla of each semicircular canal; the hair-cells are grouped in a ridge or crest known as the *crista.* The sense-organs then of the kinetic-static labyrinth, instead of consisting of a continuous organ as in the cochlea, are definitely five in number—the macula of the saccule, the macula of the utricle, and the three cristæ of the three semicircular canals. The macula of the saccule is an oval spot extending across the floor of the saccule in the frontal plane of the head. The macula of the utricle extends in the antero-posterior or sagittal plane of the head. The two maculæ, therefore, are at right angles to each other.

Overlying each of these end-organs is a gelatinous substance; in the cochlea it is found overlying the units of the Organ of Corti and is known as the *tectorial membrane.* Over the macula of the saccule and also over the macula of the utricle there extends a so-called *otolith membrane,* which contains tiny calcareous nodules known as otoliths. The hairs of the crista of each semicircular canal are likewise surrounded by a gelatinous substance known as the *cupula.*

Briefly then, the end-organ of the cochlea is the Organ of Corti; that of the saccule, as also of the utricle, is the macula; whereas that of each semicircular canal is the crista. The hair-cells of these various sense-organs are the origin of all the peripheral filaments of the VIII Nerve. The filaments from the saccule and the filaments from the posterior semicircular canals unite to form one bundle which enters Scarpa's ganglion in the internal auditory canal. The filaments from the utricle unite with those from the superior and horizontal canals to form another bundle which also continues to Scarpa's ganglion. Scarpa's ganglion therefore contains all the fibres from the kinetic-static labyrinth. The VIII Nerve itself consists of two distinct bundles, a cochlear portion and a vestibular portion, united, however, within one neurilemma. The Nerve is approximately one-half inch long and extends from the internal auditory canal to its entrance into the brain-stem at the junction of the medulla oblongata and pons, where it again breaks up into its respective cochlear and vestibular portions.

## PHYSIOLOGIC CONSIDERATIONS

Physiologically the internal ear may be divided into three portions—the *acoustic* labyrinth concerned exclusively with the function of hearing, the *static* labyrinth, for the maintenance of station, and the *kinetic* labyrinth, for the recognition and analysis of motion. The entire portion of the internal ear concerned in maintaining equilibrium has heretofore been called the *static* labyrinth; this organ, however, has a much broader use than merely a static function. As Randall points out, there is this third function, which he has termed "kinetic." The acoustic function is limited to the cochlea, the static function is limited to the saccule and utricle, whereas the kinetic function is presided over by the three semicircular canals and also probably by the saccule and utricle. When the body is at rest the otoliths by their pressure on the maculæ of the saccule and utricle give information as to the position of the body; this, accurately speaking, is the static function. The kinetic function is dependent upon the entire equilibratory portion of the ear; the three semicircular canals take cognizance of rotary movements of the body in all conceivable planes. Movements in a linear direction, anteroposteriorly, are probably detected by the macula of the utricle; linear movements in a lateral direction are appreciated by the macula of the saccule; and linear movements in a vertical direction, up and down, are detected by both the utricle and saccule.

The underlying principle of the physiology of the labyrinth is that primarily the end-organ is a hair-cell stimulated by wave-impulses. These impulses are then conducted by means of a nerve-filament from the hair-cell. The hairs are set in motion by waves of the endolymph. In the organ of Corti the hair-cells are set in motion by sound-waves conducted to the cochlea, either by air or bone. Wave-impulses in the external ear cause a movement of the drum-head; these impulses are carried mainly by the chain of ossicles through the foot-plate of the stapes, which vibrates in the oval window, causing similar wave-like movements in the endolymph. These in turn impinge upon the hairs of the organ of Corti. Sound-waves may also be transmitted to the endolymph more directly by bone-conduction; but the essential feature is that the stimuli to the organ of Corti, producing the appreciation of sound, originate in the external world. On the other hand, in sharp contrast, the hair-cells of the kinetic labyrinth are usually set in motion by the movement of the body itself and not by an external wave-impulse. If the body moves forward in a linear direction there results a lagging behind of the otoliths above the macula of the utricle. If the body moves sideways there is a lagging behind of the otoliths above the macula of the saccule. A rotary movement of the body causes movement of the endolymph in one or more of the semicircular canals, thereby affecting certain of the hairs in the corresponding crista or cristæ. When the current of the endolymph is toward the ampulla, the hairs of that side are put upon the stretch; if the current is away from the ampulla the hairs of the opposite side of the crista are put upon the stretch. The hair-cells of the two sides of the crista are connected by separate nerve-fibres with different central nuclei and because of this arrangement it is possible for the cells of one side of the crista to produce diametrically opposite phenomena from the stimulation of the cells of the other side of the same crista.

Figure 7 shows the lumen of the horizontal membranous canal; the bulging portion of the outer end is the ampulla and from the external portion projects the crista. It has been proven experimentally that a current toward the ampulla in the horizontal canal produces twice as strong a reaction as a current away from the ampulla. This is represented diagrammatically by two hairs on one side of the crista and one hair on the other side. Figure 8 represents the current away from the ampulla; it will be noted that the one hair is therefore put upon the stretch, whereas the two hairs on the opposite side are relaxed. In Figure 9, on the other hand, with the current toward the ampulla, the two hairs are put upon the stretch, whereas the one hair on the opposite side is relaxed. Figure 10 represents the vertical canals, the superior and the posterior, uniting in one common crus. In the vertical canals the sense-organs are so constructed that the current away from the ampulla is twice as powerful as the current toward the

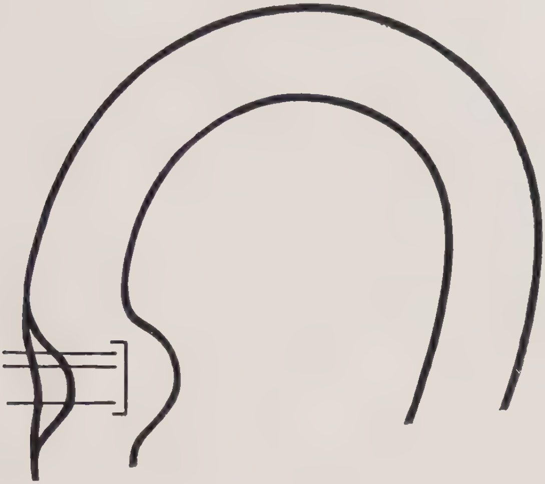


FIG. 7.—Scheme of the right horizontal canal.

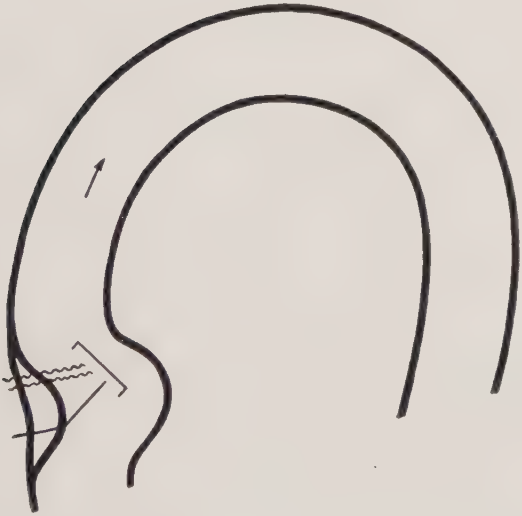


FIG. 8.—Current away from the ampulla in the right horizontal canal. (Only one-third of the hairs are put upon the stretch.)

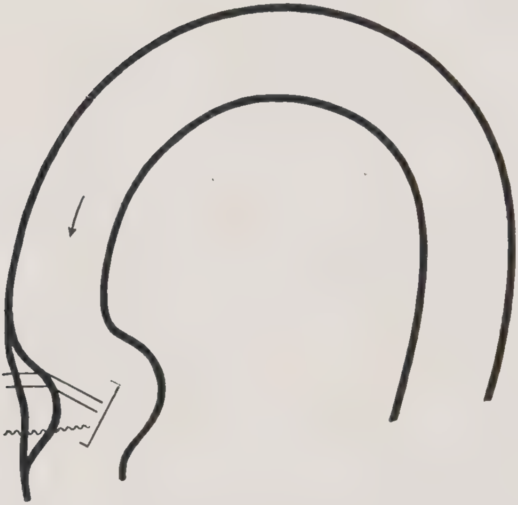


FIG. 9.—Current toward the ampulla in the right horizontal canal. (Two-thirds of the hairs are put upon the stretch.)

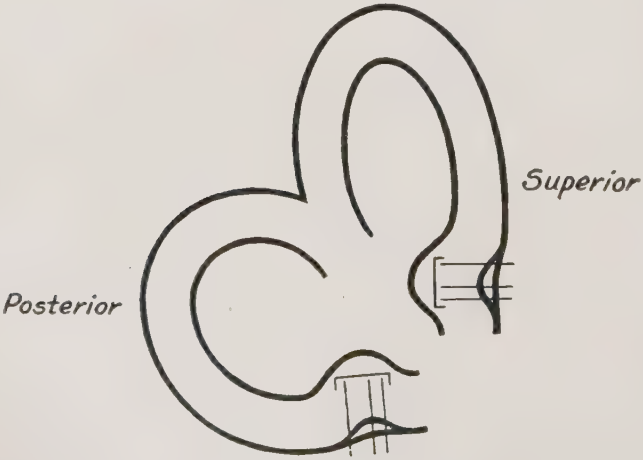


FIG. 10.—The vertical semicircular canals (right).

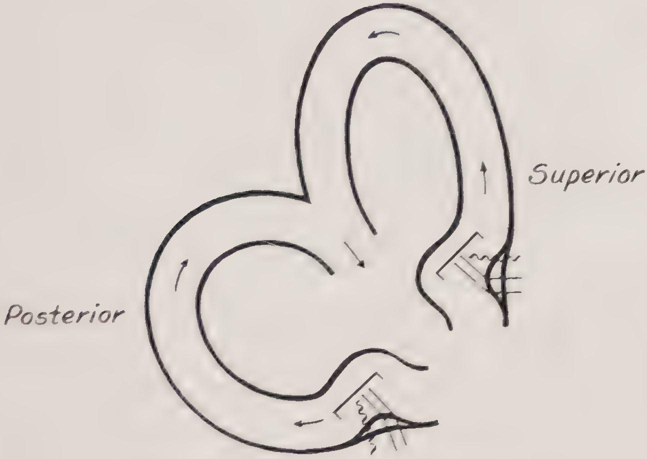


FIG. 11.—Current away from the ampullæ.

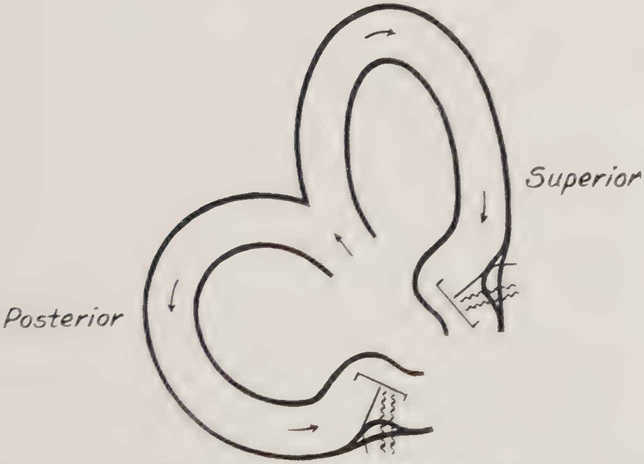


FIG. 12.—Current toward the ampullæ.

ampulla—just the opposite from the mechanism of the horizontal canal. Figure 11 shows that the current away from the ampullæ puts the two hairs of each crista upon the stretch, whereas the one hair is relaxed. Figure 12 shows that the current towards the ampullæ puts only one hair of each crista upon the stretch, whereas the other two in each ampulla are relaxed. It will be noted that the current either away from or toward the ampullæ of the vertical canals has an identical effect upon both the superior and the posterior canal mechanism.

It is true that the actual calibre of the membranous labyrinth is so minute that it is inconceivable to consider a free circulation of the fluid within it. It may be as Neumann suggests, the actual movement occurs in the perilymph inducing molecular movement in the endolymph. It is not necessary, however, that there should be a free movement within the endolymph, as even the slightest impulse can appreciably affect the sensitive hairs projecting into the lumen of the semicircular canals. We do know, clinically, that the only explanation of the actual phenomena we observe is that there is a movement of the endolymph. Barany has shown that douching the ear with the head forward produces exactly the opposite responses from douching the ear with the head backward. The necessity of considering that there is an endolymph movement may be proved as follows: Incline the head backward 60°; this puts the horizontal canal in the vertical position. Douching the right ear with cold water then produces (1) horizontal nystagmus to the left, (2) sensation of rolling to the left, (3) past-pointing to the right, and (4) a rolling or falling to the right.

Now without any further douching, merely tilt the head forward 120°, which puts the horizontal canal in the vertical plane, but exactly upside down from its previous position; there immediately appear (1) horizontal nystagmus to the *right*, (2) sensation of rolling to the *right*, (3) past-pointing to the *left*, and (4) a rolling or falling to the *left*.

This, of course, disposes absolutely of the theory that cold water causes a depression and hot water causes a stimulation of the hair-cells of the crista. Merely by altering the position of the head there are produced diametrically opposite phenomena, and a movement of the endolymph is the only explanation for this clinical fact.

Whereas anatomically each semicircular canal is actually little more than a half-circle, physiologically it may best be regarded as a circular canal; so far as the movement of the endolymph is concerned a complete circle is made through the utricle which unites the two ends of the anatomically *semi*-circular canal.

In considering the physiology of the internal ear, one is necessarily impressed with the curious fact that two organs of such diversely different functions—the organ of hearing and the organ of balance—should be housed in one common cavity. The anatomy of these two organs of utterly different functions is almost identical. There is not only a continuous bony capsule within which this double organ is contained, but the actual membranous internal ear itself is one continuous sac, containing the endolymph which has a direct communication from the farthermost point of the cochlea anteriorly to the farthermost point of the semicircular canals posteriorly. The endolymph itself is the same in the cochlea, the saccule and utricle and the canals. The actual nerve-mechanism is very similar—practically identical; the auditory unit of the cochlea is analogous to that of the macula of the vestibular sacs and of the crista of each semicircular canal. Furthermore, on all of these structures are cells with projecting hairs. Again, at the end of these hairs, in each instance there is a membrane-like substance—the tectorial membrane over the organ of Corti, the otolith-membrane over each macula, and the cupula over each crista.

In spite of the almost identical anatomical construction of these various sense-organs, they have widely different functions. Why this should be may be explained by considering the development of the internal ear as seen in various lower forms of life. The equilibratory organ was one of the earliest special senses and made its appearance almost coincidently with life itself. This primitive sense-organ depended entirely for its impulses upon shocks and concussions occurring in its environment. The sense of hearing, also dependent upon external wave-impulses, was an outgrowth of this function. As W. A. Wells suggests, there was already developed an organ for the reception of wave-impulses and nature had but to add an annex with a specialized nerve-mechanism adapted to the perception of vibrations of a certain periodicity, which becoming fused are appreciated as sounds.

A possible connecting link between the sense of equilibration and hearing, as seen in the human internal ear, is suggested by Bonnier in the following functions: (1) Baresthesia. By this is meant the appreciation by the saccule and the utricle of the pressure exerted by external media whether liquid or air. (2) Seisesthesia. By this is meant the appreciation by the utricle and saccule of abrupt variations of the pressure from external media. These two functions are neither hearing, on the one hand, nor the sense of motion or station on the other. The ability of the saccule and the utricle to recognize pressure from external media might well be regarded as an intermediary function between the sense of balance and the sense of hearing.

All of the impulses from the various end-organs within the internal ear are conducted by the respective nerve-filaments, which, after leaving the labyrinth, form the VIII Nerve and continue to their respective cells or centres. After reaching the brain-stem at the junction of the medulla oblongata and pons. these impulses, until then confined within the VIII Nerve, travel along widely divergent paths.

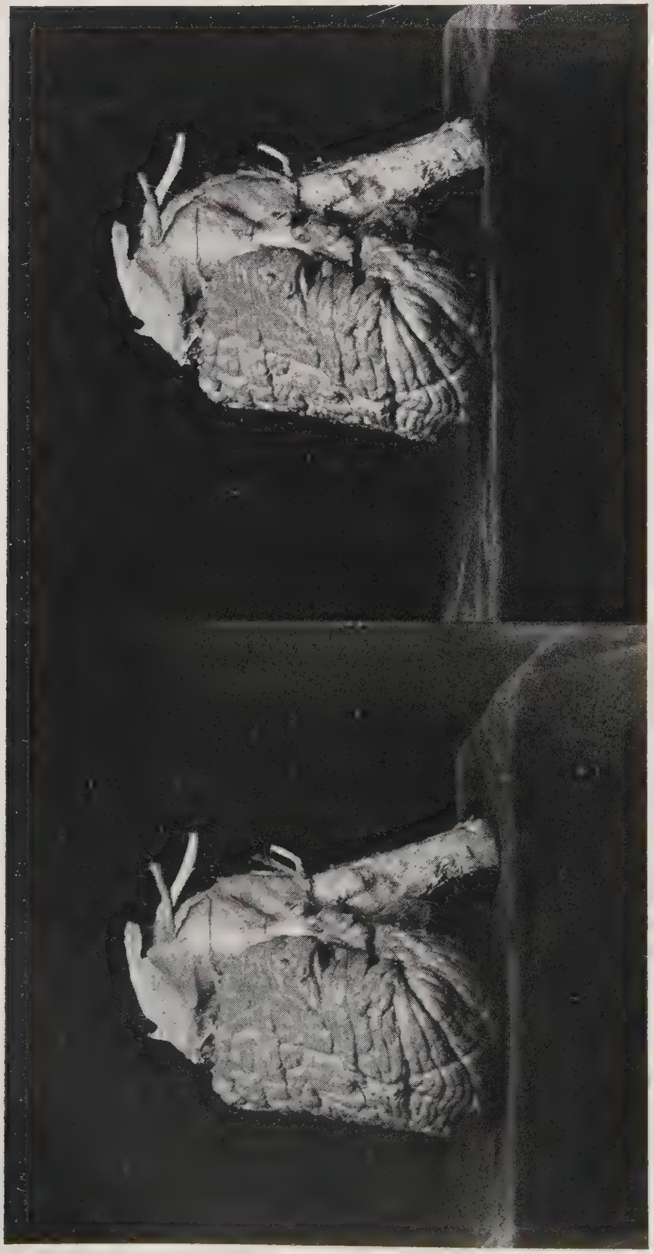
## CHAPTER XII

## MEDULLA OBLONGATA, PONS AND CEREBELLUM

The stereoscopic photographs shown as illustrations for this chapter were made from dissections of the various structures concerned in this study—the VIII Nerve, medulla oblongata, pons, the three pairs of cerebellar peduncles, the cerebellum, the cerebral crura and the posterior portion of the first and second convolutions of the temporal lobe. It is within these structures that are contained all the nerve-pathways from the ear.

The vestibular fibres of the VIII Nerve pass through the medulla oblongata and pons on the way to the cerebellum. For clinical purposes it is convenient to speak of the medulla oblongata and the pons as one structure—the brain-stem (Figs. 13, 14 and 15). The brain-stem commences at the upper termination of the spinal cord and ends at the upper portion of the pons where it divides and continues as the two cerebral crura. The lower portion of the brain-stem is the medulla oblongata (Fig. 15). It is one to one and one-fourth inches long and resembles a somewhat flattened cone. Its apex or lower end is continuous with the spinal cord, while the upper, broader extremity is attached to the pons. Anteriorly, it rests upon the upper surface of the baso-occipital bone and shows several prominences. The two swellings immediately alongside the median anterior fissure are the anterior pyramids. Outside of these are two other swellings corresponding to the two olivary bodies. The upper portion of the posterior surface forms a triangular area and is the floor of the lower part of the IV ventricle (Fig. 14).

The substance of the medulla oblongata is made up of longitudinal and curving nerve fibres (most of them the continuation of nerve tracts from the spinal cord), interspersed with masses of gray matter or nuclei. It is of interest to note that nuclei of the cranial nerves from the VIII to the XII inclusive are found within the medulla oblongata. In its upper portion there are also



G. 13.—View of right side of spinal cord, medulla oblongata, pons and cerebellum.

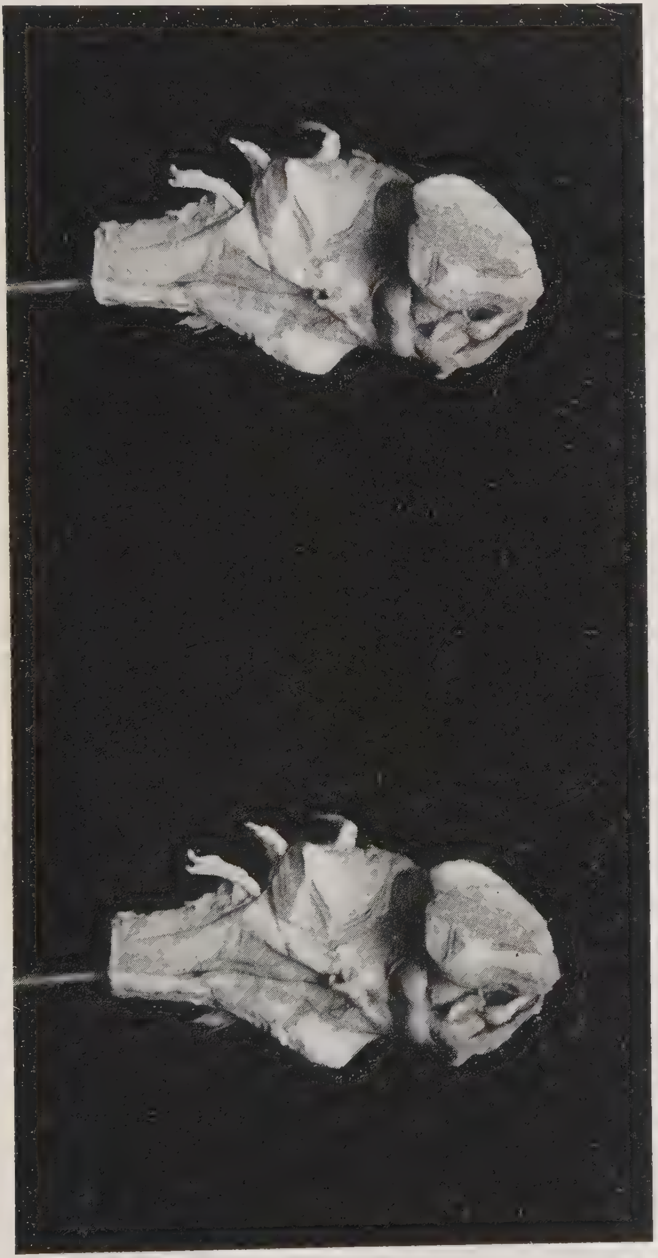


Fig. 14.—Left side of brain-stem, viewed from behind and above.

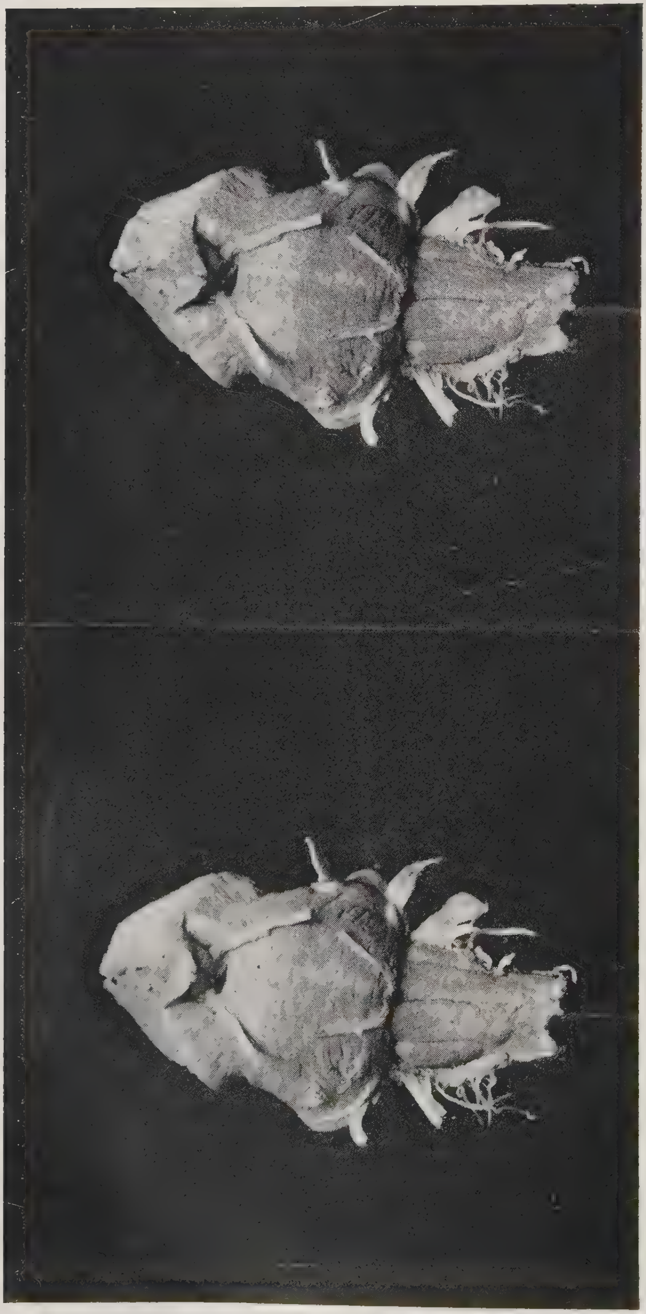


Fig. 15.—Brain-stem viewed from in front and below.

found three collections of nerve-cells constituting the Deiters' nucleus, the triangular nucleus and Von Bechterew's nucleus. These nuclei receive some of the vestibular fibres from the VIII Nerve after it enters the brain-stem.

The pons or the upper portion of the brain-stem (Fig. 15) lies in the posterior cranial fossa between the medulla oblongata below and the crura cerebri above. It is a cuboidal mass of tissue measuring about an inch from before backward, a little over an inch in length and about an inch and a half from side to side. The anterior surface of the pons consists entirely of transverse fibres arranged in three groups—upper, middle and lower. These pass to the lateral surfaces which in turn continue backwards and outwards to become the middle peduncles of the cerebellum.

The median portion of the posterior surface of the pons (Figs. 14, 16, 17) forms the upper part of the floor of the IV ventricle. Among other anatomical markings can be seen the acoustic tubercle which extends downward to the medulla oblongata, while crossing it are the uppermost of the acoustic striæ, which run from behind the juxtarestiform body across the floor of the IV ventricle and disappear in the median sulcus. The posterior surface of the pons, where it forms the floor of the IV ventricle, consists of gray matter in which are found the dorsal nuclei of the VIII Nerve, the nuclei of the VI Nerve and the sensory and motor nuclei of the V Nerve.

The inner structure of the pons is made up largely of masses of fibres both longitudinal and transverse, among which are interspersed numerous small collections of gray matter known as the nuclei pontis. The longitudinal fibres are mainly continuations of the pyramidal tracts running from the cerebrum to the spinal cord. Of the transverse fibres there is one commissural tract uniting the two lobes of the cerebellum, while the other two are decussating tracts. One of these runs from the cerebellum, decussates and terminates in the opposite side of the pons; the other transverse tracts also decussate but do not originate in the cerebellum. They start from the nuclei pontis. From the opposite side of the pons where these two sets of transverse tracts terminate,

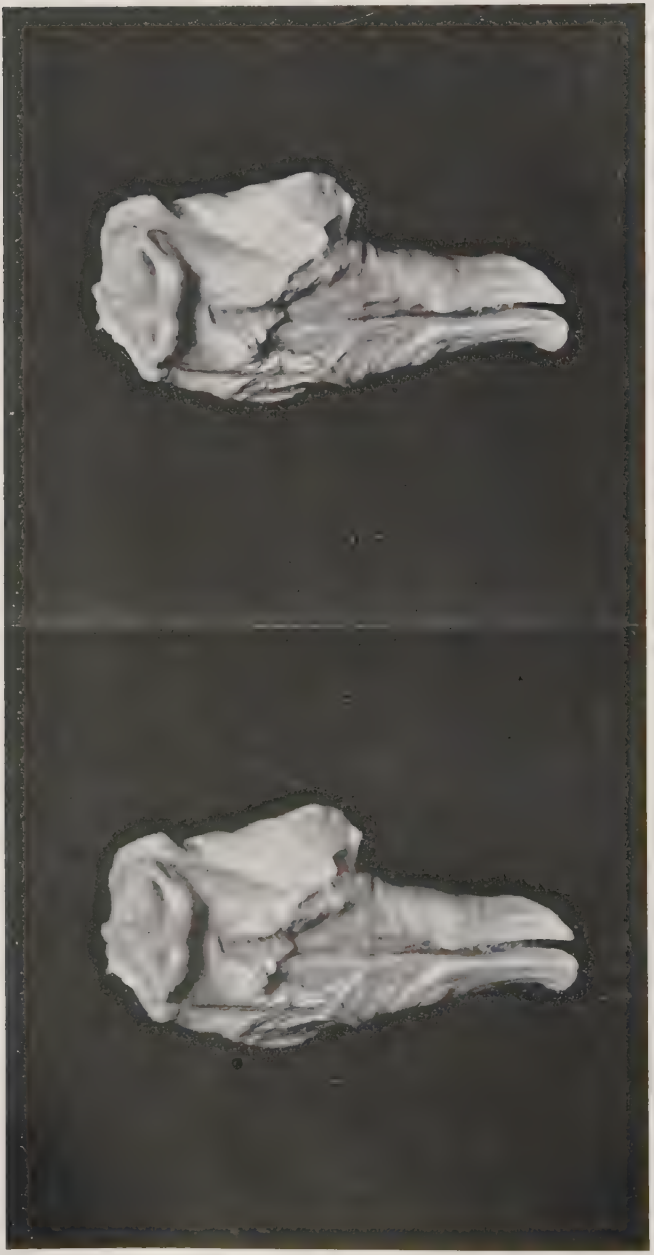


FIG. 16.—Medulla oblongata and pons viewed from behind and above, showing the acoustic striæ in the floor of the IV Ventricle. Also shows cerebellar peduncles.

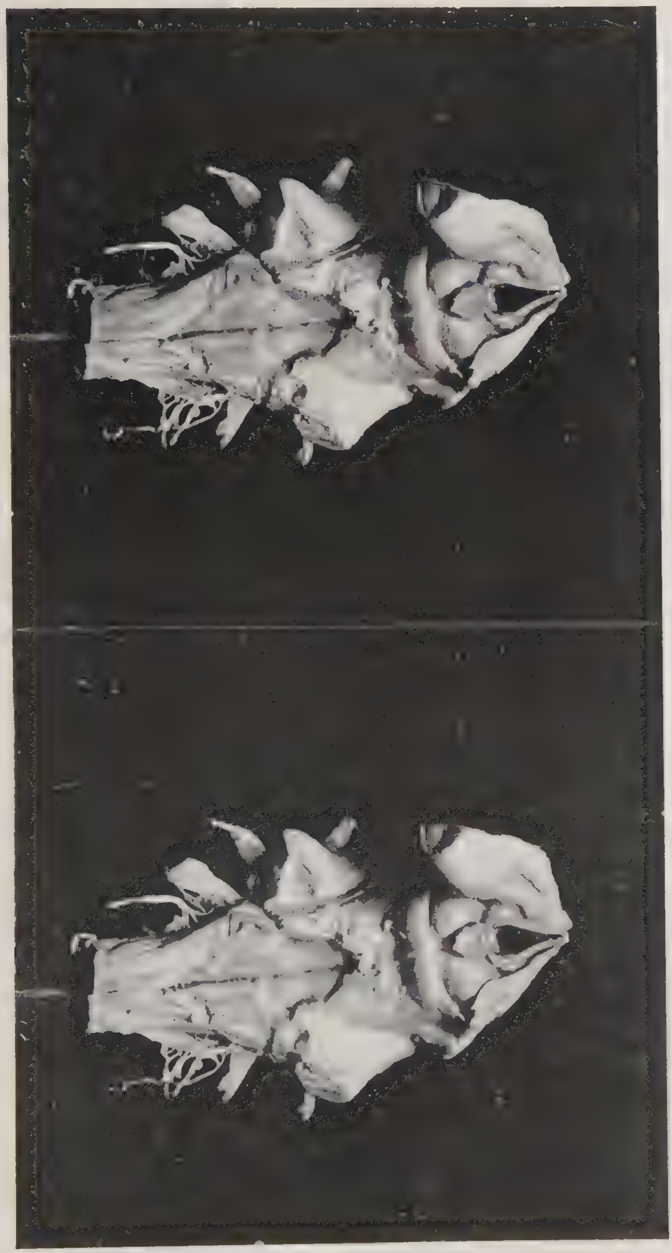


FIG. 17.—Medulla oblongata, pons, cerebellar peduncles and crura cerebri, viewed from behind and above. Shows boundaries of IV Ventricle.

the fibres continue upward to the cerebrum. In this way the cerebellum is connected by nerve-tracts with nuclei in both sides of the pons.

All the cranial nuclei concerned in nystagmus are located in the brain-stem and for that reason are of particular interest in this study. The nuclei of the III and IV cranial nerves are located in the crura cerebri low enough to be in relation with the upper portion of the pons. In the lower portion of the pons is found the only other nucleus concerned in eye-movement, the VI, or abducens. When speaking of the location of the cranial nerves in relation to the brain-stem, it is well to bear in mind that the cranial nerves have both a deep origin and a superficial exit. The exit of the cranial nerves from the III to the XII are all to be found on the lateral and ventral aspects of the pons and medulla oblongata. The origin of these nerves is to be found on the posterior aspect of the pons and medulla oblongata and in close relation with the floors of the III and the IV ventricles.

In order that lesions may involve the "cell mass" of the cranial nerves, they must be situated in the extreme dorsal portions of the pons and medulla oblongata; root fibres, however, may be attacked by lesions situated anywhere in the brain-stem. It might be remarked, in passing, that the VI cranial pair of nerves, the abducens, are very slender structures and traverse a rather lengthy course outside of the brain-stem. For that reason affections anywhere within the cranium are very likely to affect them by pressure, with resulting paralysis or paresis. The VII and VIII cranial nerves are very close to each other, almost in one common sheath. It is also well to note that the VIII Nerve is very much softer than its neighbor, the VII Nerve, so that when both are involved in one lesion it is not at all uncommon to find a complete loss of function of the VIII Nerve, whereas the VII or facial nerve is only slightly affected because of its greater toughness and power of resistance.

The nerves of the greatest interest in our study are the III cranial nerves or motor oculi, as well as the IV and VI pairs of cranial nerves, since these are concerned in nystagmus. The X nuclei are of interest because of the associating nausea and vomiting which often result from stimulation of the other cranial nerves through reflex sources. To make it possible for the III, IV and VI cranial nerves to work in harmony there must necessarily be a connecting link of fibres. This is furnished by the posterior longitudinal bundles which are located in the posterior portion of the crura, pons and medulla oblongata. These bundles of fibres not only connect the ocular cranial nuclei with each other, but constitute the anatomic structure which makes it possible for all of the cranial nerves to work in co-ordination. For example, when a person looks to the right he not only uses the right external rectus muscle, but also the left internal rectus, and there must also be a normal antagonistic action of the other ocular muscles. In other words, there must be a normal co-ordination of the right VI and left III nuclei, and to a lesser extent of the other nuclei controlling the ocular muscles. At the lower portion of the medulla oblongata, the posterior longitudinal bundles become continuous with the fibres of the lateral limiting zone and with the fibres of the antero-lateral ground bundle, which probably play the same part in the cord that the posterior longitudinal bundles play in the brain. The posterior longitudinal bundles are seen in the pons and medulla oblongata as two triangular bundles, one on either side of the central raphé; they are placed immediately below the gray matter of the floor of the IV ventricle. Motor fibres in the posterior longitudinal bundles, which start from the nucleus of the III Nerve, descend to the pons, where they join the VI Nerve of the same side. Fibres which arise in the nucleus of the VI Nerve of one side ascend, cross the middle line, and join the fibres of the III Nerve of the opposite side, along which they pass to the opposite internal rectus muscle, thus making possible co-ordinated movements.

The IV ventricle is a rhomboidal, lozenge- or diamond-shaped space. It is situated between the brain-stem anteriorly and the cerebellum posteriorly (Figs. 14, 16, 17). Its lower part lies in the interior of the medulla oblongata, while the upper portion is situated between the pons in front and the cerebellum behind. Its anterior boundary is the so-called floor of the IV ventricle. The lower extremity of the ventricle is continuous with the central canal of the spinal cord, and the upper end is continuous with the Aqueduct of Sylvius. The lateral boundaries from above downward on each side are formed by the superior peduncle of the cerebellum, the middle peduncle, the restiform body (or inferior peduncle), and the fasciculus gracilis; the upper part of the anterior boundary of the floor is formed by the pons, while the lower part of the floor is formed by the medulla oblongata. A median sulcus divides the floor into two longitudinal halves. On each side of this shallow sulcus is a longitudinal ridge known as the fasciculus teres. In the lower part of the floor these two fasciculi become smaller and form together a pointed process known as the calamus scriptorius. All of the auditory fibres cross at this calamus scriptorius so that a lesion there could destroy audition on both sides. The roof of the IV ventricle is formed in its upper portion by the two superior cerebellar peduncles, in its middle portion by the vermis of the cerebellum into which it extends like a slit, and in the lower portion by the posterior wall of the medulla oblongata.

The cerebellum (Figs. 13, 18, 19, 20, 21) lies in the posterior fossa of the cranium behind the pons, immediately below a fold of membrane called the tentorium cerebelli, which separates it from the cerebrum above. Its structure is made up of an outer layer of gray matter surrounding a core of white matter in which are imbedded several gray nuclei, among them the three vestibular nuclei, globosis, emboliformis and fastigii, the so-called roof nuclei, which receive the vestibular fibres from the labyrinth. The cerebellum (Figs. 22 and 23) is made up of two lateral lobes usually called hemispheres, connected by a median portion known as the vermis. The vermis of the cerebellum is the most primitive portion and is the only lobe present in fishes, reptiles and birds. The cerebellum is connected with the brain-stem by three pairs of peduncles (Figs. 22, 24), the two superior cerebellar peduncles above, the two inferior cerebellar peduncles below, and the two middle cerebellar peduncles between the other two pairs. Each cerebellar hemisphere is divided into a superior and an inferior portion; it is also anatomically divided into lobules, but for all

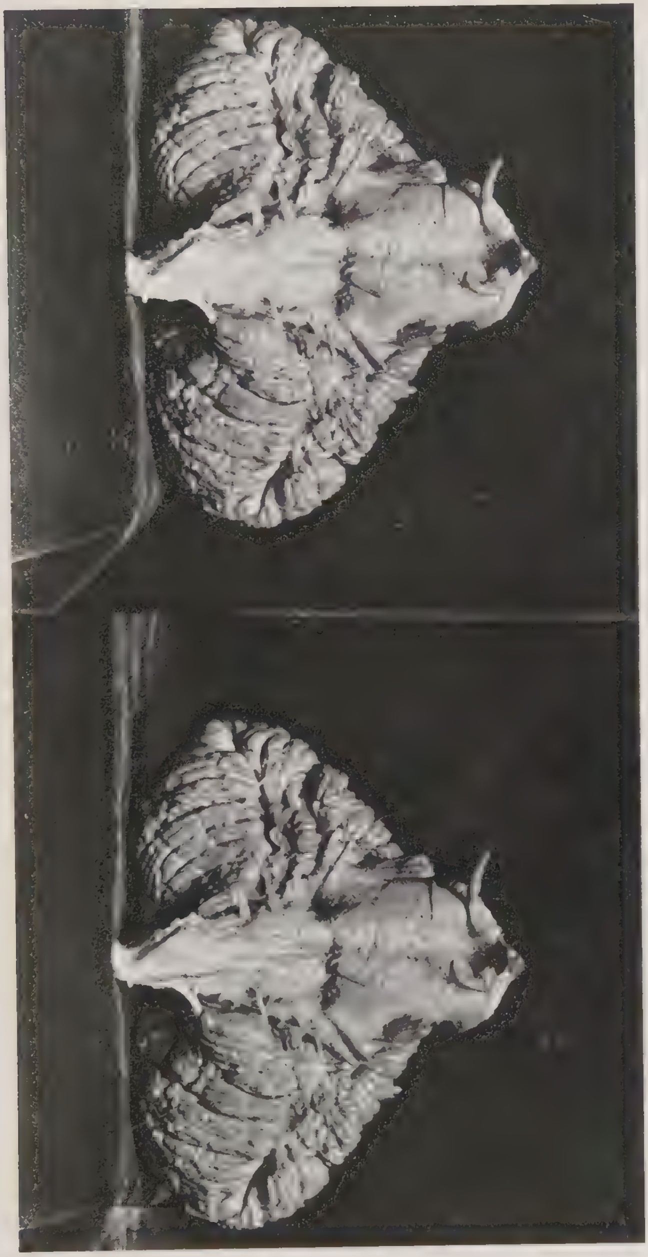


Fig. 18.—Cerebellum and brain-stem viewed from below.

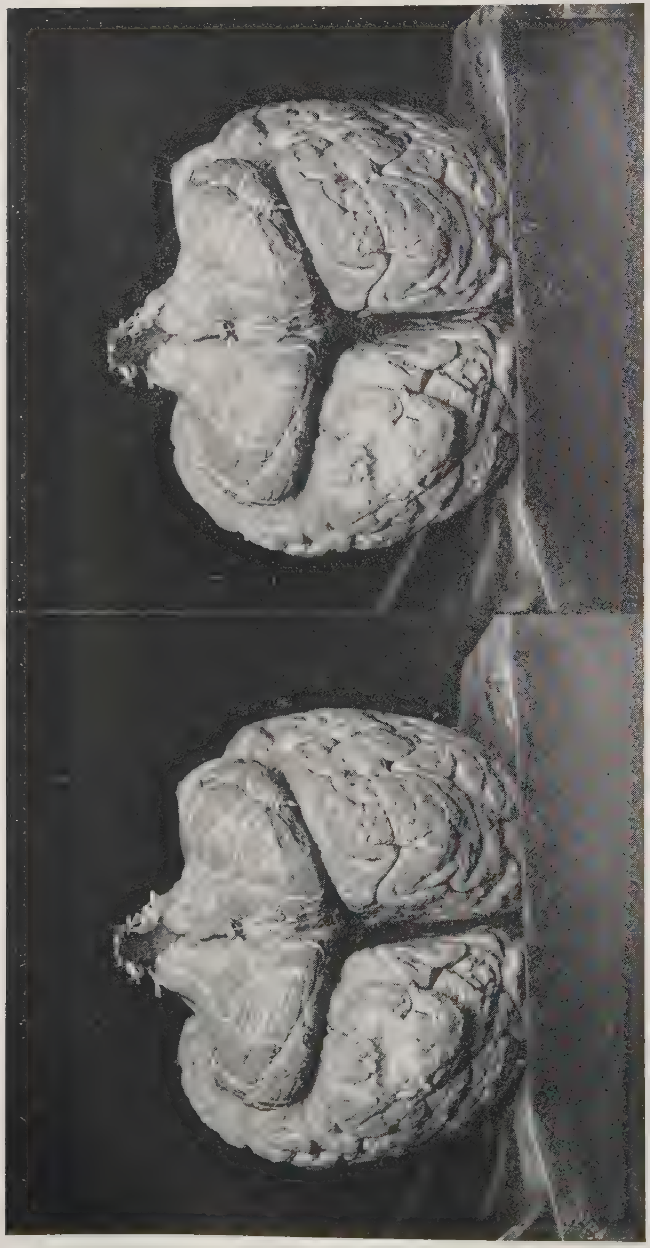


Fig. 19.—Posterior view of relation of cerebrum and cerebellum.

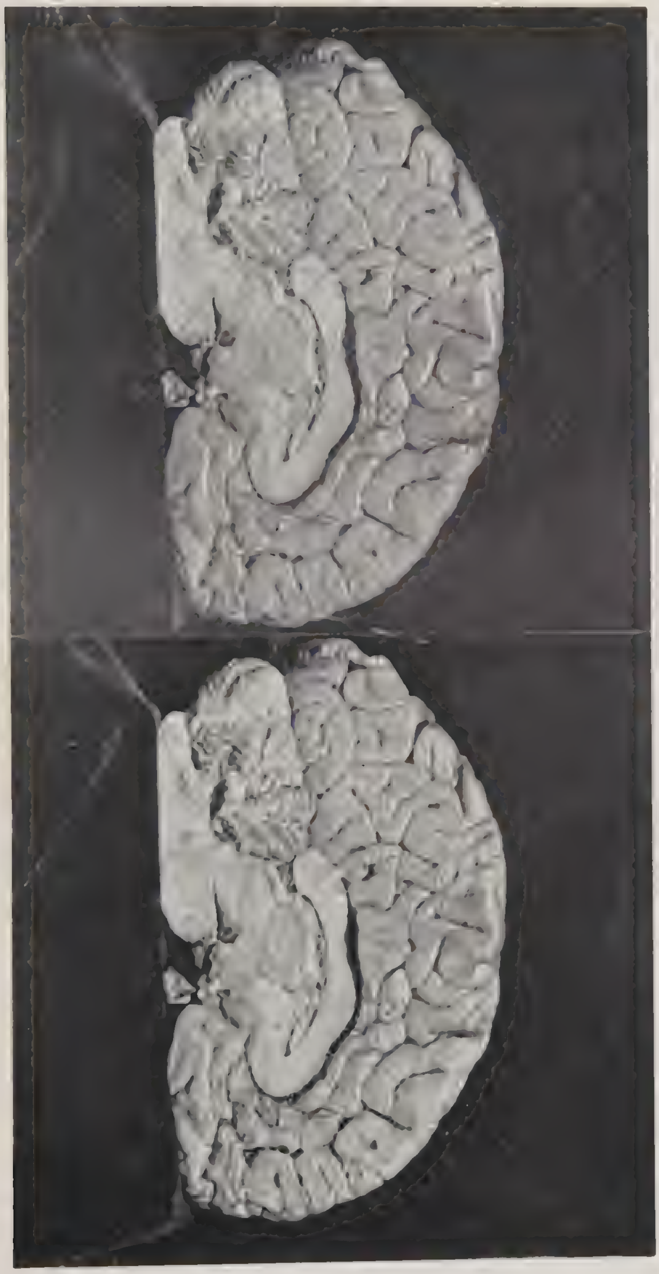


Fig. 20. Sagittal section of brain, showing entire left half of cerebrum, pons, medulla oblongata and cerebellum

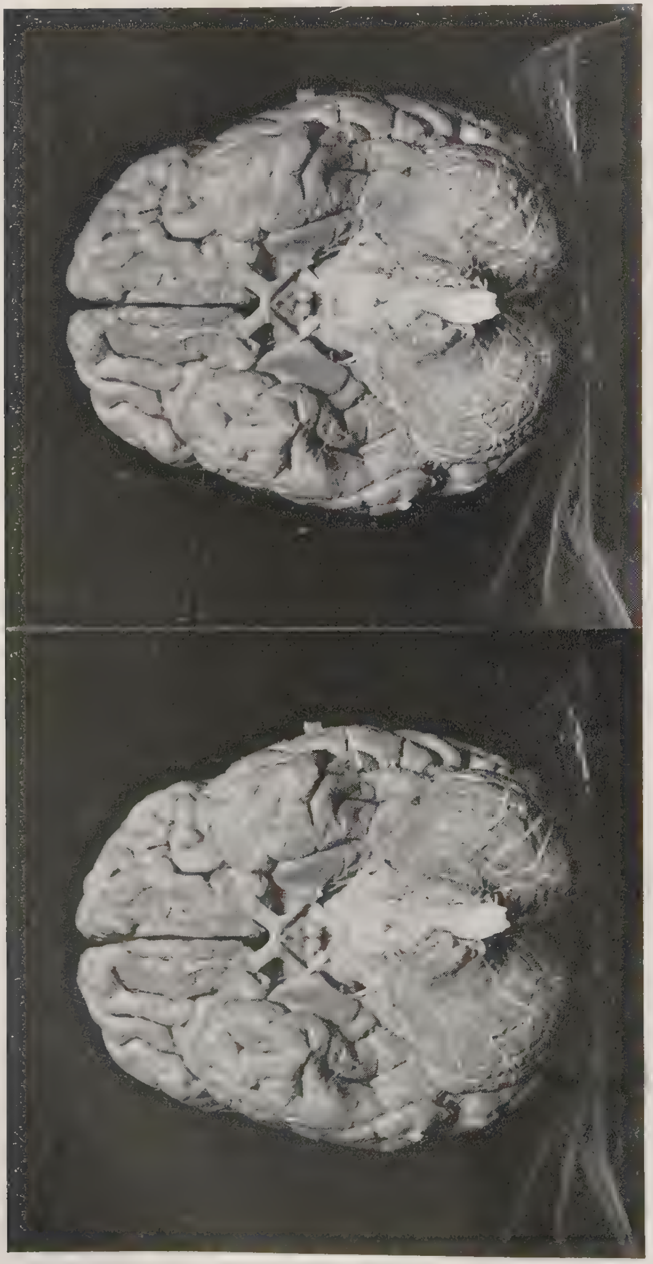


FIG. 21.—Inferior aspect of cerebrum, brain-stem and cerebellum.

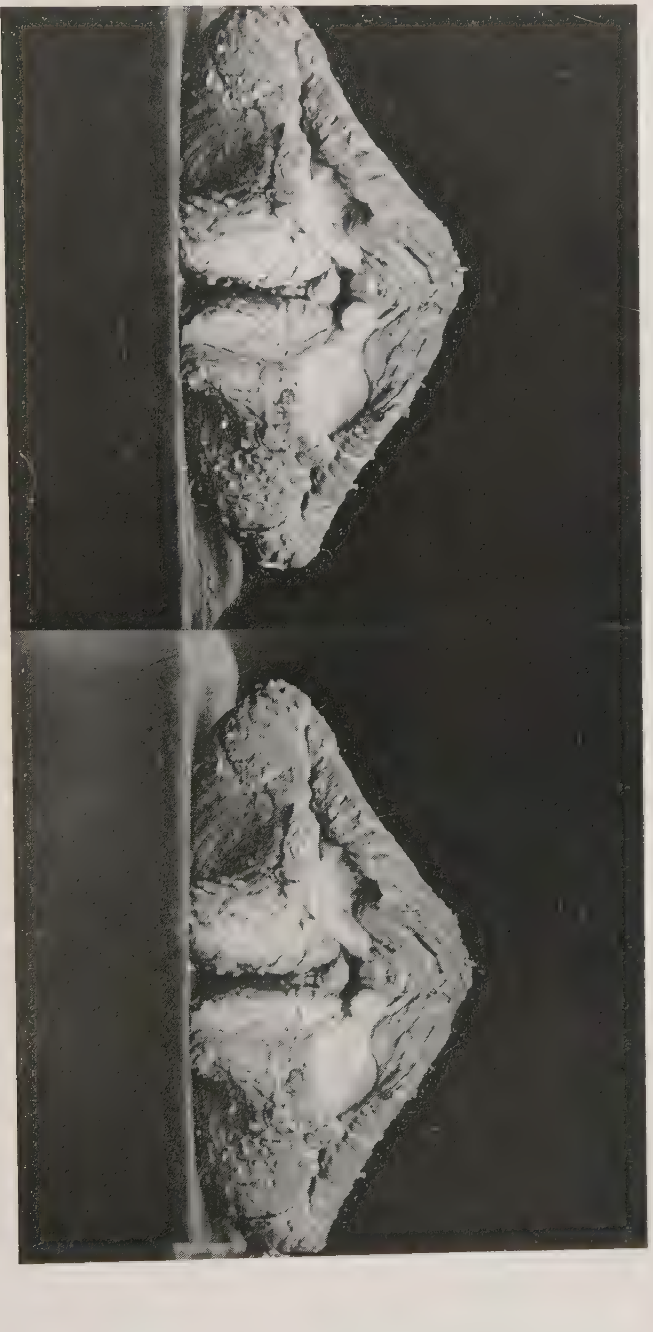
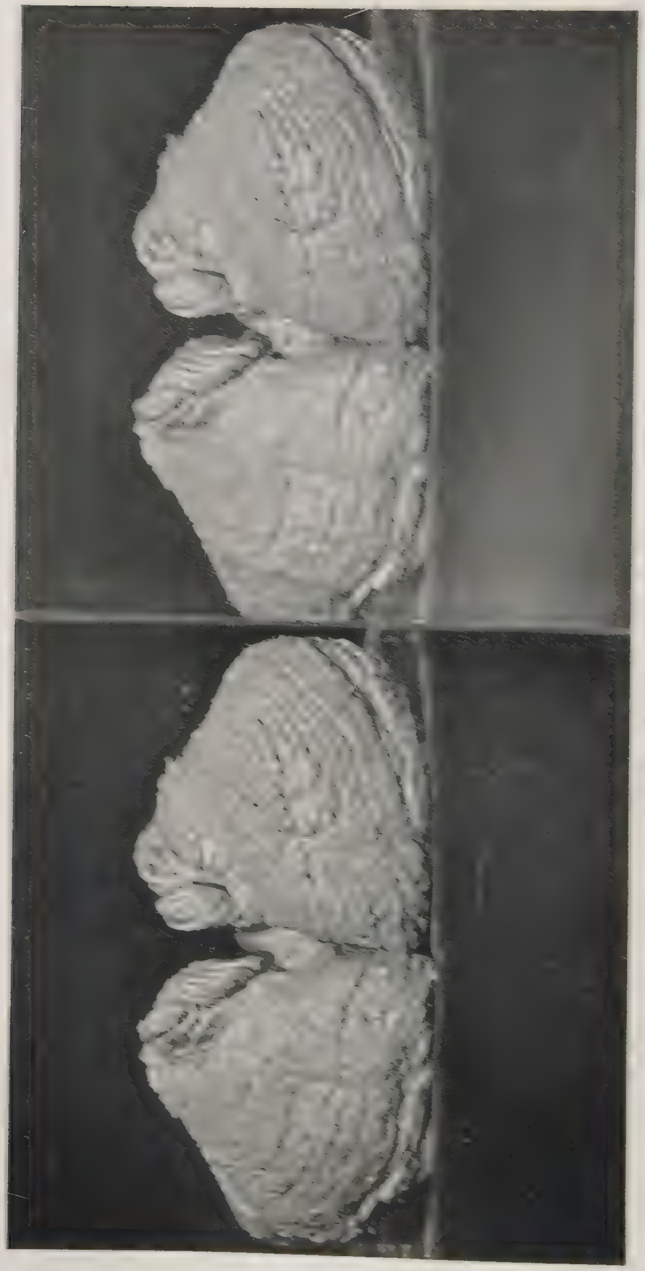


FIG. 22.—Cerebellum viewed from below and in front. Shows cerebellar peduncles.



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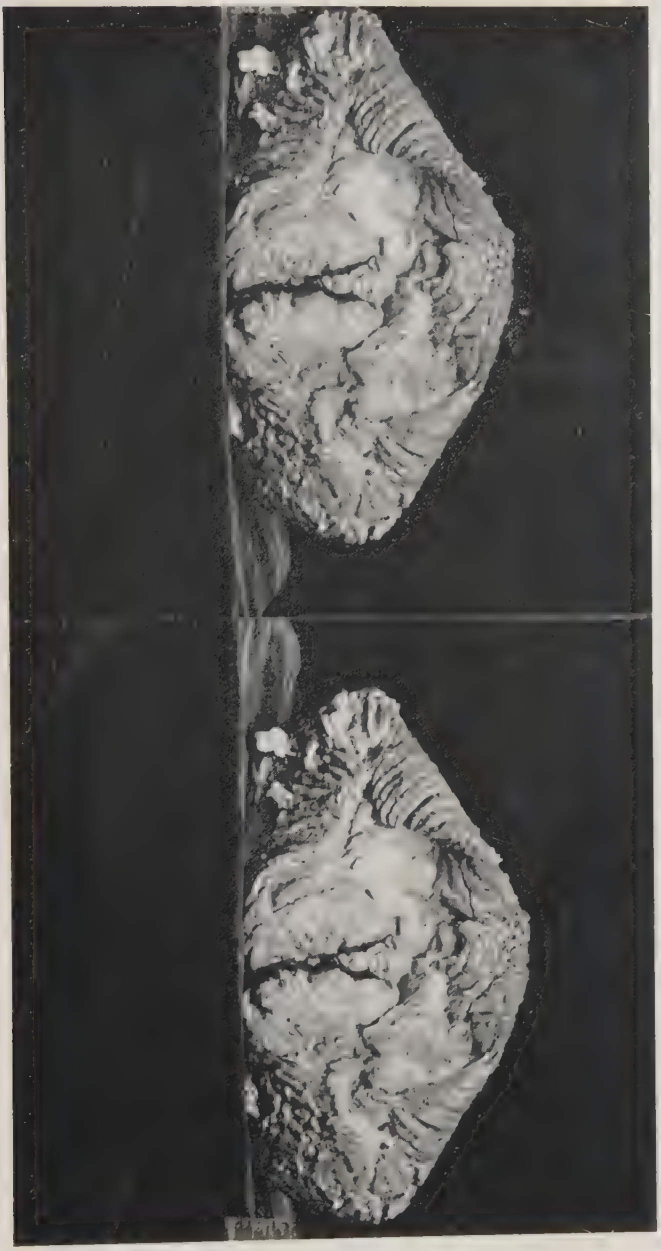


Fig. 24.—Cerebellum viewed from below and in front. Shows cerebellar peduncles.

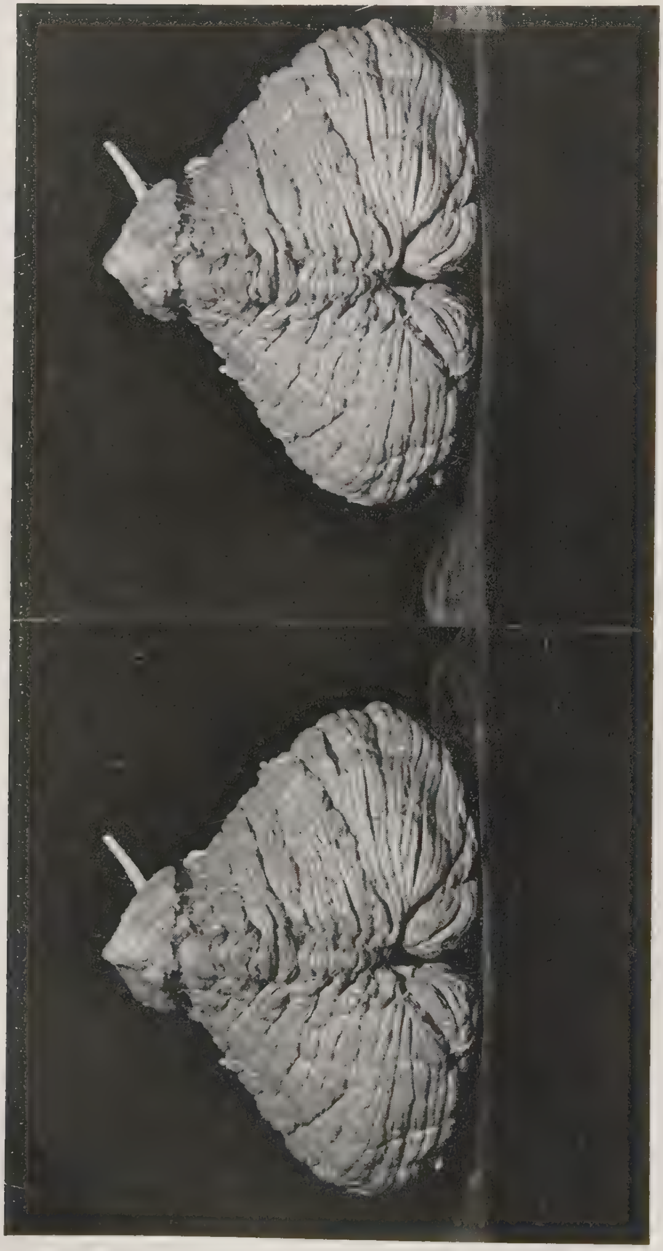


Fig. 25.—Cerebellum and cerebral crura viewed from above and behind.

practical clinical purposes, especially in reference to localization, it is sufficient to consider each hemisphere as divided into a superior and inferior portion. The vermis has also been subdivided into various portions, but for our purposes it is sufficient to consider the vermis to consist of a superior and an inferior portion, since it is these portions of the vermis that are supposed to contain within them distinctly different functionating centers.

The cortex of the cerebellum is composed of large cells from which nerve-fibres originate. Some of these fibre tracts are connected with a central group of cells within the cerebellar hemisphere known as the dentate nucleus. Other fibre tracts coming from either the cerebellar cortex or from the dentate nucleus are in connection with either the cerebrum, pons or spinal cord. The inferior cerebellar peduncles or restiform bodies connect the anterior border of the cerebellum with the medulla oblongata. They pass upward and inward toward the vermis of the cerebellum and the upper part of the lateral lobes. They consist chiefly of fibres which come from the spinal cord and contain the vestibular fibres which come from the horizontal semicircular canals of the labyrinth.

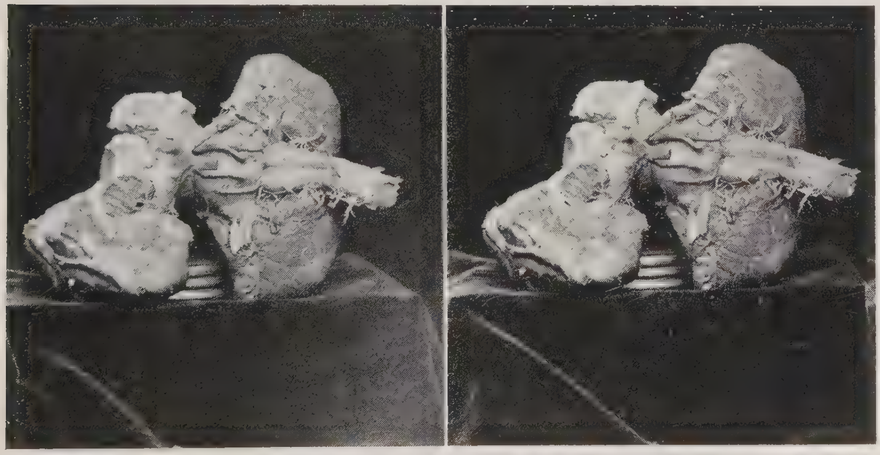


FIG. 26.—Dissection of structures through which impulses producing vertigo pass to the posterior portion of first and second temporal convolutions.

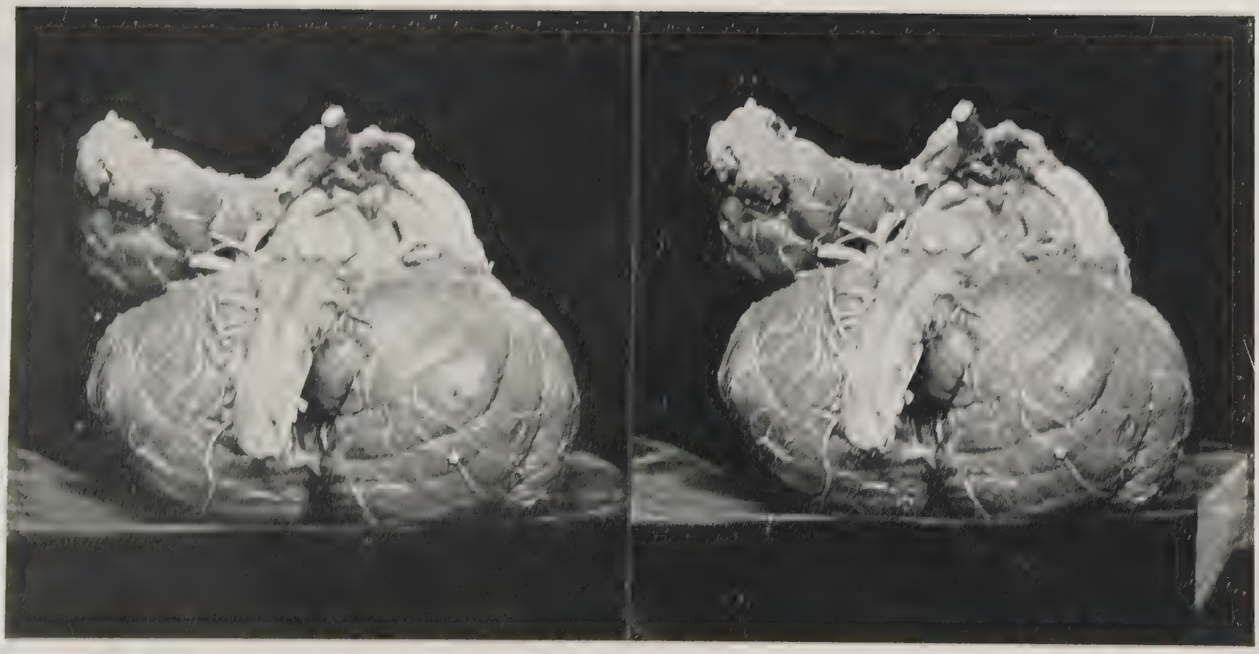


Fig. 27.—Dissection of structures through which impulses producing vertigo pass to the posterior portion of first and second temporal convolutions.

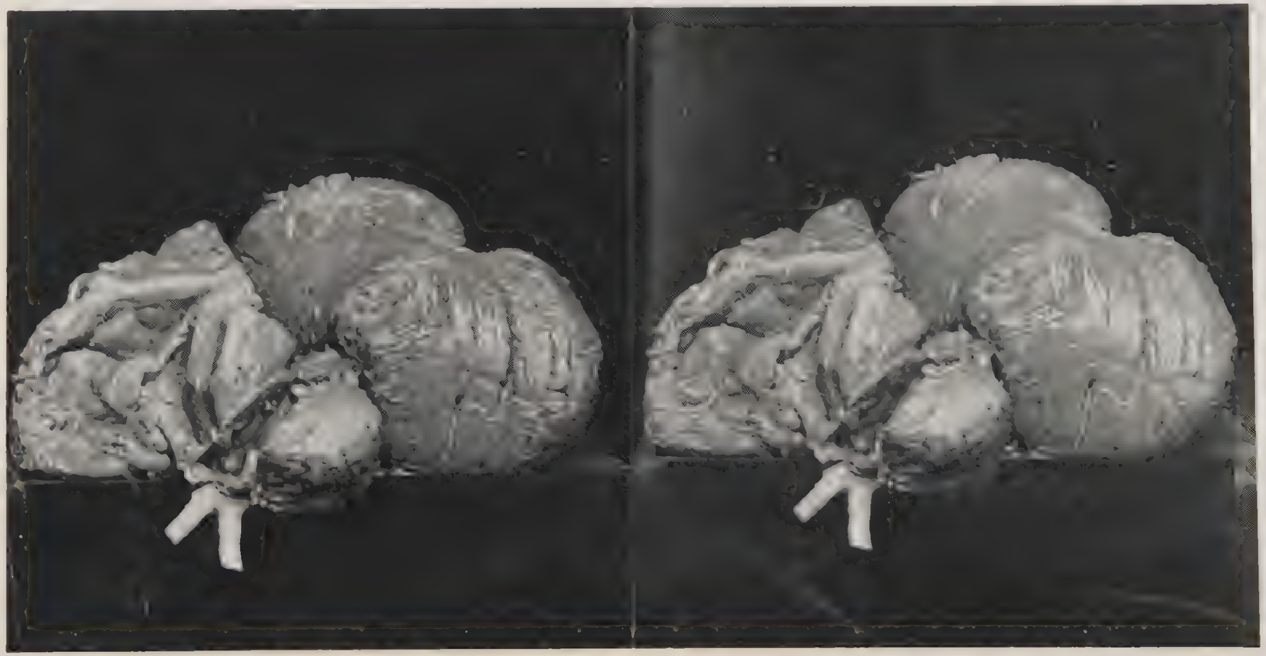


Fig. 28.—Dissection of structures through which impulses producing vertigo pass to the temporal lobe

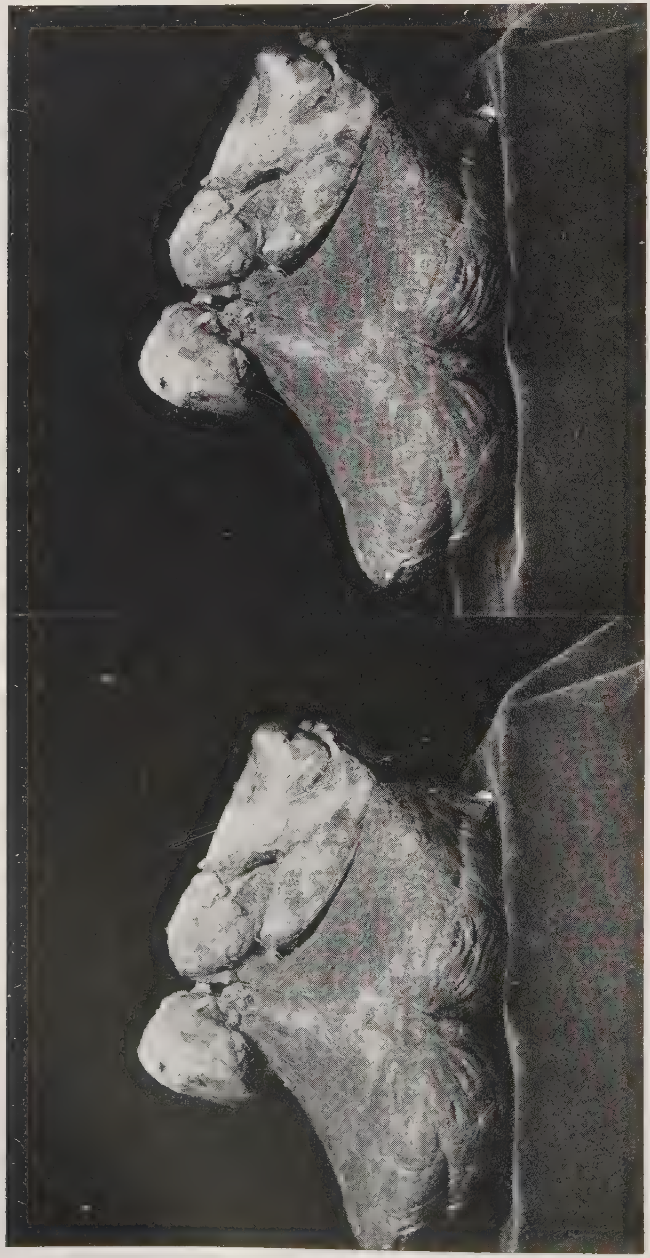


FIG. 29.—Dissection of structures through which impulses producing vertigo pass to the temporal lobe.

The middle peduncles, the largest of the cerebellar peduncles, pass backward from the pons toward the cerebellum and are found to the outer side of the inferior cerebellar peduncles. They consist almost entirely of the transverse fibres from the ventral portion of the pons. These fibres connect the cerebellum with the pons, the fibres ending in a group of cells in the pons, on the side opposite to their origin. From here a new system starts and goes to the internal portion of the foot of the cerebral peduncles and then into the frontal lobes. The middle cerebellar peduncles also probably carry the vestibular fibres from the labyrinth which originate in the vertical semicircular canals.

The superior cerebellar peduncles are small bands of white matter which emerge from the anterior border of the cerebellum, run forward and inward at the sides of the upper part of the IV ventricle, to the upper part of the pons. Each superior cerebellar peduncle connects the cerebellar hemisphere of that side with the cerebral hemisphere of the opposite side, the fibres decussating in the upper portion of the pons, then entering the cells of the red nucleus in the posterior portion of the cerebral crura. From here new groups of fibres arise and enter the optic thalamus from which they are distributed to the lateral portions of the cerebral hemisphere of that side.

We do not possess correct knowledge of the function of all the fibres of the different cerebellar peduncles. We do know, however, that the inferior cerebellar peduncle is concerned with the synergic movement of the limbs, and also contains vestibular fibres from the horizontal semicircular canal. The middle cerebellar peduncles are probably concerned with synergy of the trunk, and contain the vestibular fibres from the vertical semicircular canals of the labyrinth, while the superior cerebellar peduncles are probably concerned with co-ordinating the motor and sensory impulses of the cerebral cortex with those of the cerebellum.

# CHAPTER XIII

## CEREBELLAR LOCALIZATION

The views on cerebellar localization and cerebellar symptomatology presented in this chapter have been adapted from the localization advanced by Mills and Weisenburg. It must be understood that the scheme of localization in the human cerebellar cortex advanced by them is only tentative and in some particulars is merely suggestive.

The cerebellum is essentially a motor organ and is concerned with supplying the necessary synergy for bodily movements. As bodily movements are as a rule complex movements, it is evident that no one centre or single area representing any one part could well represent the entire bodily movement in question. The eyes may move to the right or left or up or down, without association of any other movements, but on the other hand, the eyes may move synergically with the head or with the head and trunk or with the head and trunk and limbs. Similarly the trunk may be bent forward or backward, twisted to the right or left, with little or no association with movements of the extremities. Such comparatively simple trunkal movements, however, are not the rule in daily life. The rôle played by the cerebellum has its best exemplification in such acts as advancing and retiring in the erect position or in the performance of complex movements when standing, as in fencing, boxing or public speaking; or in driving and regulating a rapidly moving automobile; or in any highly trained movements such as are exhibited in a series ascending in complexity and rapidity of association from a game of tennis or baseball to the handling of an aeroplane.

No *one* area in the cerebellum could represent such a complexly associated neuromechanism. In other words, cerebellar localization is compound in its cortical representation. A composite, however, has always within it factors or elements which can to a less or greater extent be separated from the entire mass. In the human cerebellum we must be able to recognize a cortical representation for synergic movements whether simple or complex. Each simple synergic movement has, however, its special cortical representation. Cerebellar cortical representation is analogous to cerebral cortical localization, but only analogous and not similar. In the cerebral cortex each individual movement is definitely represented by a group of cells, which, under the control of the will, initiates the necessary energy that results in the contraction of the particular muscle group necessary for that movement. Such a cortical area supplies energy "in the rough"—energy pure and simple and unguided. In order to carry out a definite movement of a limb or any portion of the body, more than one muscle has to contract at the same time to produce the desired result. The muscles concerned in the particular movement derive their energy from the cerebral cortex; this energy, however, must be guided or synergized in order that this movement be performed with any degree of precision and nicety. Such a guide for this energy is furnished by the cerebellum.

The cerebellum therefore is not concerned with the contraction of any one muscle but rather with groups of muscles, which, in turn, are concerned in certain definite movements. Let us take, for example, the shoulder joint. There is a certain group of muscles which always act when the upper extremity moves outward. There is likewise another group of muscles which act when the extremity moves inward. and similarly a group of muscles for moving it upward, and still another for moving it downward. While each one of these movements is individually represented in the cerebral cortex, in the cerebellar cortex all these movements are represented in an outward-pointing centre, an inward-pointing centre, an upward-pointing centre and a downward-pointing centre.

When an individual desires to move his upper extremity downward and touch a certain point, the cerebral cortex supplies the energy necessary for the muscles to move the arm downward. This alone, however, while supplying the energy necessary for the movement would not result in a very precise movement. The necessary precision is furnished by the cerebellum. In this particular movement the centres concerned would be the outward-pointing centre and the inward-pointing centre for that extremity. These two centres would act against each other so that the arm would be brought down in a perfectly straight line. Should the individual desire to touch a certain point with his right upper extremity, moving it from side to side, the cerebellar control would be manifested by a functioning of the upward-pointing centre and downward-pointing centre for that extremity.

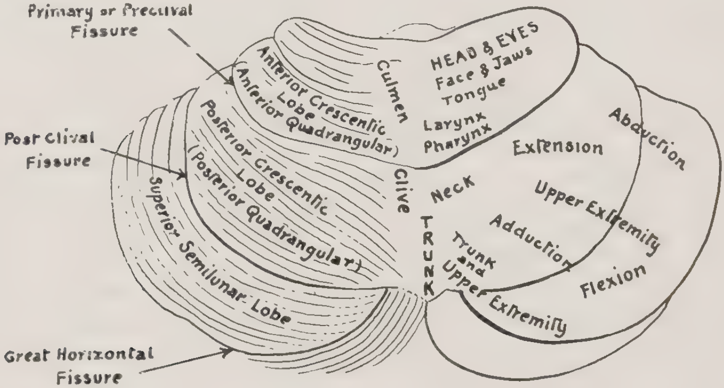


Fig. 30.—Cerebellar localization; zones and centres of the superior surface of the cerebellum.

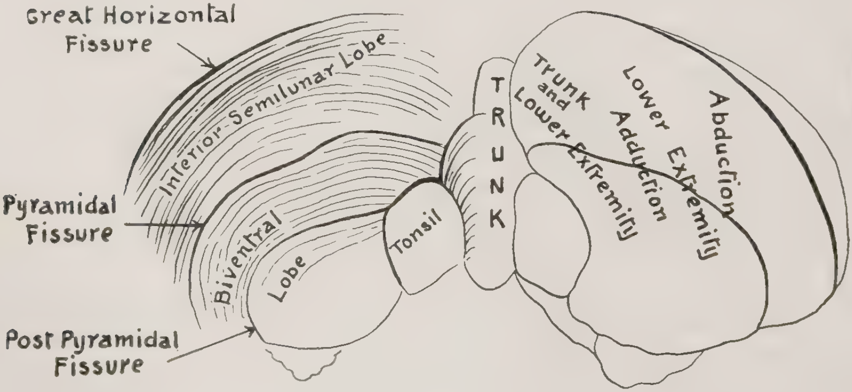


Fig. 31.—Cerebellar localization; zones and centres of the inferior surface of the cerebellum.

Unfortunately the exact location of the centres for the different simple movements of the various joints is not yet known. Cerebellar cortical representation is believed to be homolateral, the right side of the cerebellum supplying synergy for the right side of the body. Based on studies of cerebellar embryology, comparative anatomy, experimentation and clinical pathology, a tentative scheme of cerebellar localization was worked out as shown in figures 30 and 31. In connection with this work on neuro-otology, we take advantage of this scheme in attempting to throw light on the location of cerebellar lesions. The outward-pointing centre for the shoulder is believed to be located in the anterior portion of the quadrangular lobes, whereas the centre for the inward-pointing of the right shoulder is located in the posterior portion of the quadrangular lobe, of the right cerebellar hemisphere.

In this connection we must note that Barany conducted experiments on the living human being, on whom extensive cerebellar decompressions had been done. By means of ethyl chloride he chilled certain areas of the cerebellar cortex and then by vestibular stimulation noticed whether there was any deviation from the normal past-pointing. When such ear-stimulation was applied that the right arm should past-point to the right, he noted that such past-pointing was absent when he had chilled the superior and inferior semilunar lobes of the right cerebellar hemisphere. He further noted that when he chilled the biventral lobe of the right cerebellar hemisphere, the right arm could not be made to past-point inward after such stimulation that should make it past-point inward. He therefore concluded that the outward pointing centre for the shoulder joint is located in the semilunar lobe and that the inward pointing centre of the shoulder is located in the biventral lobe.

From a clinico-pathologic standpoint, when stimulation, no matter how applied, fails to produce a past-pointing—let us say of the right arm to the right—it would suggest some disturbance of the outward-pointing centre in the right cerebellar hemisphere. Likewise when stimulation fails to make the right arm past-point to the left, it would suggest a disturbance of the inward-pointing centre of the right cerebellar hemisphere.

Clinically speaking, the thought to bear in mind is that the cerebellum is concerned with synergy for all the movements of the body and whatever symptoms may be present, such as hypermetry, adiadokokinesis or tremor, all result from loss of synergy.

The vermis of the cerebellum governs the pelvic girdle muscles and a lesion of the vermis is suggested if, for example, on pushing the patient backward the pelvis does not move forward as it should normally. In such cases the patient may fall directly backward like a broomstick.

In presenting the symptomatology of lesions of special portions of the cerebellum and its connections, we first discuss lesions confined strictly to the cerebellum; second, lesions implicating the superior, middle and inferior cerebellar peduncles; third, lesions of the cerebello-pontile angle; and, last, lesions invading the cerebello-pontile angle secondarily.

We desire to emphasize that, to make an accurate diagnosis of a cerebellar lesion, it is necessary to take into consideration not only the cerebellar symptoms, but all other symptoms. We have been repeatedly impressed with this fact since our attention has been more especially directed to this subject. Often we have made a diagnosis of a lesion of the labyrinth only to find the cerebellum implicated, and vice versa; again we have diagnosed cerebello-pontile-angle tumors only to find that the angle was secondarily invaded by a lesion growing from the cerebellum. It has not been infrequent to diagnose lesions of the cerebellum when the tumor has been in the third ventricle and has involved the superior cerebellar peduncles secondarily.

## Diagnosis of Lesions Strictly Confined to the Cerebellum.

In accordance with our views of cerebellar function, lesions of the cerebellum itself cause more strictly limited symptoms than those which invade any of the peduncles. Most tumors of the cerebellum are gliomatous and are of slow growth. The majority tend to invade the middle rather than the outer portions of the cerebellum, and as a consequence, the vermis is nearly always involved wholly or in part, in addition to one of the lateral lobes. We believe that in the superior vermis are centred the synergic movements of the upper trunk or shoulder girdle and in the inferior vermis the movements of the lower trunk or pelvic girdle. As a consequence there will always be in such lesions, in addition to disturbances in the movement of one or both limbs, alterations in the station and gait. In cases in which the vermis is implicated, the staggering is mainly forward or backward, rather than to one side, and of such cases, it is more marked in the shoulder girdle type. In our experience, if in addition to the involvement of the vermis, the lateral lobes are implicated, the sway of the trunk is to the side of the lesion, although it must not be forgotten that in a progressive cerebellar disease the patient may learn, for example, to go voluntarily to the left to offset his staggering to the right.

In those instances in which the lateral lobes alone are implicated, the asynergic movements are present only in the limbs of the involved side; if in the superior lobe, in the upper limb, and if in the inferior lobe, in the lower limb. By direct stimulation of the vestibular apparatus by the turning and caloric tests it is possible to obtain, for example, a total lack of outward pointing or inward pointing; this is not so readily demonstrated by other tests.

General Symptoms.—The presence of cranial nerve symptoms, as of the VII, VI, V, IX, X or XII, indicates that the lesion is extracerebellar. Involvement of the motor fibres, with paresis of the limbs, increased reflexes and Babinski, indicates that there is pressure on the motor apparatus and should not be expected in lesions strictly confined to the cerebellum, except in those cases in which the lesion is very large. As a rule, motor involvement indicates a pontile lesion or a lesion in the angle pressing on the pons. Headache, nausea, vomiting and choked disc are, of course, early manifestations, but only in those instances in which there is interruption in the flow of the cerebro-spinal fluid; they generally indicate a lesion either in the crus or pons pressing on the aqueduct of Sylvius.

## Lesions of the Cerebellar Peduncles

We possess little definite knowledge of the functions of the fibres in the cerebellar peduncles. It is supposed that the inferior and middle peduncles transmit impulses to the cerebellum, and that the superior peduncle transmits impulses from this organ. It is probable, however, that all the peduncles transmit impulses in both directions. It is also probable that the inferior peduncles are concerned chiefly with synergic movement of the lower limbs, and to a lesser degree of the upper limbs, the middle peduncle with the synergic movement of the trunk and head, and that the superior peduncles are mainly concerned with the association of cerebral with cerebellar functions. All this, however, is theory, based somewhat on anatomic and physiologic facts, but not as yet supported by pathologic and clinical data.

In the literature there are a few instances of lesions confined more or less to one of the peduncles, but it cannot be said that our clinical experience is such that we can differentiate the asynergy resulting from lesions of the superior from those of the middle or inferior peduncles. From our present knowledge all we can say is that lesions strictly confined to any of the peduncles cause asynergic symptoms in all parts of the body and as a consequence we are compelled to make the diagnosis not on the cerebellar symptoms but on those associated with them.

There are instances in the literature in which so-called turning or rolling movements have been recorded, in tumors of the middle and of the inferior peduncles, either toward or away from the side of the lesion; but these cases are not definite and cannot be relied on. There is, however, this difference in the cerebellar symptomatology of the different peduncles. Because of the anatomic relation of the superior peduncles, lesions of this part usually invade both sides, the cerebellar symptoms being present all over the body, and only rarely, as in a case reported by Mills in which there was thrombosis of the superior cerebellar artery, is the asynergy limited to the limbs on one side. As the middle cerebellar peduncles are more widely separated, such lesions are often unilateral, and tumors implicating the inferior cerebellar peduncle are almost always one-sided.

### Lesions of the Cerebello-pontile Angle

The diagnosis of tumor in this area as a rule is not difficult. We have encountered cases, however, in which such diagnosis had been made, only to find that this angle was invaded secondarily by tumors growing from the cerebellum and more rarely from the pons. The differential diagnosis is important from the surgical standpoint, for it is readily seen that tumors growing from the pons or cerebellum offer little hope for surgical removal.

Tumors growing in this angle are usually fibromatous and more rarely fibrosarcoma. We have encountered a number of cysts, but these are generally parts of gliomatous tumors growing from the cerebellum. Most tumors grow from the VIII and more rarely from the VII, V, or VI cranial nerves respectively. The first symptoms are usually referred to the cranial nerve from which the tumor grows and in the course of time the other nerves in the angle become implicated. Of course, the symptoms depend on the size of the growth. In small tumors no pressure is exerted on either the pons or cerebellum. In a well-developed case, besides the cranial nerve symptoms, because of the pressure on the pons, there are motor symptoms on the opposite side of the body and more rarely sensory phenomena.

Because of the pressure on the anterior part of the cerebellum, both the superior and inferior surfaces are involved, this causing cerebellar asynergy of both limbs of the same side. In cases in which the angle is invaded secondarily from the cerebellum or from the pons, the cerebellar symptoms are always much more marked, and it is because we have not been sufficiently careful in the differentiation of the cerebellar phenomena that errors in diagnosis have arisen.

## CHAPTER XIV

# TRACTS OF THE AUDITORY APPARATUS AND OF THE VESTIBULAR APPARATUS

Having up to this chapter discussed the anatomy and physiology of the labyrinth, the VIII Nerve, the medulla oblongata, the pons and the cerebellum, we now consider those tracts or paths which, as it were, link them together. It is generally known that there must exist a definite nerve-pathway between the cochlea and the cerebral cortex in order that stimulation of the cochlea by sound-waves shall produce the conscious sensation of hearing. Similarly, if stimulation of the semicircular canals causes the eyes to move, it is evident that there must be a nerve-path between the ear and the eye muscles. Further, if stimulation of the semicircular canals produces a conscious sensation which we call vertigo, it is evident that there must be a nerve-path between the ear and that portion of the cerebral cortex which receives this impulse. The course of the auditory fibres has been histologically demonstrated by many investigators so that the pathways along which sound impulses travel are more or less known; not so with the vestibular apparatus. The recognition of the ear as the chief equilibratory organ is so recent that most of its intracranial pathways are still undetermined. On the basis of over 700 clinical cases, including a considerable number of operations and autopsies, we have come to certain conclusions in regard to these pathways, some of them definite and others needing further analysis.

Ramon y Cajal has shown histologically that fibres from the vestibular portion of the VIII Nerve enter Deiters' nucleus and continue through the inferior cerebellar peduncle into the cerebellum itself. This path is generally recognized and accepted. Our observations up to the present time have suggested in addition the following:

(1) After entering the brain-stem the fibres from the horizontal semicircular canal have a distinct and separate pathway from the fibres from the vertical semicircular canals.

(2) The fibres from the vertical semicircular canals ascend into the pons.

(3) The fibres already demonstrated by Cajal are those from the horizontal semicircular canal exclusively.

(4) The fibres conveying impulses to the eyes are distinct and separate from those conveying the impulses producing vertigo.

(5) The fibres conveying the impulses producing vertigo go through the cerebellum en route to the cerebrum.

Our reasons for these conclusions are as follows: In 38 cases in which the labyrinths and the VIII Nerves were normal and the horizontal canal gave normal reactions, the vertical canals failed to produce the well-known responses. The following phenomena appeared: On stimulation of the horizontal canal there was obtained a normal nystagmus, vertigo and past-pointing; on stimulation of the vertical canals, however, there was *no* reaction or only a partial reaction. In some cases there was produced no nystagmus, no vertigo, no past-pointing, no falling, no nausea—in other words, all responses failed; in other cases some of the responses appeared and others did not. In some cases the vertical canals of each side failed to respond, whereas the horizontal canal of each side responded perfectly. It must be borne in mind that in all of these cases the *labyrinths* themselves were normal. In other words, the vertical semicircular canals themselves were functioning perfectly and *yet they failed to produce normal responses.* In all these cases the VIII Nerves also were normal. If, therefore, the labyrinths and VIII Nerves were normal and yet stimulation of the vertical canals failed to produce normal responses, it is evident that there is a *central* differentiation of the fibres from the horizontal canal and of the fibres from the vertical canals. Naturally this depends upon positive proof that the labyrinth and VIII Nerves were normal; this is shown by the following facts:

(1) Perfect hearing. This rendered it extremely *improbable* that there was any destruction of the vertical semicircular canals

themselves alone, leaving the horizontal canal and the cochlea entirely intact. It is similarly improbable, especially in so large a number of cases, that within the VIII Nerve those particular fibres from the vertical semicircular canals were involved, whereas the fibres from the horizontal canal and the fibres from the cochlea were absolutely intact.

(2) In each of these cases there was other corroborative evidence of involvement of the brain-stem.

(3) In five cases stimulation of the vertical canals produced no nystagmus, no vertigo, no past-pointing and no falling, and yet violent projectile *vomiting* occurred. It is evident that the vertical canals themselves and the fibres from the vertical canals in the VIII Nerve were functionating—showing, as they did, even a hyperactive response of the X nucleus to stimulation of the canals. This of course makes it absolutely conclusive that the vertical canals themselves were functionating and that the pathway was intact at least from the vertical canals to the X nucleus in the medulla oblongata. It follows, therefore, that there is necessarily a *central* differentiation of the fibres from the different canals. For detailed description of cases of this class and their analysis the reader is referred to pages 340, 347, 358, 364, 373, 390, 398, 407, 412 and 430.

In one of these cases (Mrs. D., given in detail on page 340) the vertical canals of the right ear produced no response on stimulation although the labyrinth itself, as well as the VIII Nerve, was normal. Autopsy with histological examination by Spiller showed a glioma involving the upper portion of the pons, whereas the lower half of the pons and the medulla oblongata were normal. This proves that the vertical canals fibres ascend into the pons at least as far as the location of this lesion in the upper part of the pons. Another case, declared by Weisenburg to be a classical case of lesion in the right side of the pons posteriorly in the region of the middle cerebellar peduncle, also failed in all responses on stimulation of the right vertical canals. The horizontal canal and all the canals of the left ear gave normal responses. This suggests that the vertical canals fibres were involved by this lesion in the pons on the right side. Cases in which autopsies have shown the following lesions—cerebello-pontile angle tumors pressing against the pons, intracerebellar tumors invading the pons, abscess of the IV ventricle with pressure on the pons and internal hydrocephalus with pressure within the IV ventricle upon the pons—in all of these cases stimulation of the vertical semicircular canals produced no reaction, whereas the horizontal canals gave normal responses.

If, as suggested above, lesions involving the pons cause a block in the tracts from the vertical semicircular canals, but leave the tracts from the horizontal canal intact, and if, furthermore, in these cases the medulla oblongata is proven intact, the logical conclusion is that the fibres demonstrated by Cajal in the medulla oblongata and inferior cerebellar peduncle must be the fibres from the horizontal semicircular canal exclusively. A case (Mr. L., given in detail on page 332) confirms this viewpoint. Spiller says that this patient presented the clean-cut symptom-complex generally accepted neurologically as thrombosis of the posterior inferior cerebellar artery. Spiller, H. M. Thomas, Hun and Van Gieson have demonstrated similar cases histologically; they show softening in the medulla oblongata in the region of the inferior cerebellar peduncle. In this case the lesion suggested is in the right inferior cerebellar peduncle. Ear examination showed the following: The left horizontal canal, the left vertical canals, and the right vertical canals all produced normal responses in nystagmus, vertigo, past-pointing and falling; the right horizontal canal produced a normal nystagmus, but there was a striking impairment of vertigo and past-pointing. This suggests, therefore, that the horizontal canal fibres responsible for vertigo are involved in this lesion in the inferior cerebellar peduncle. It also suggests that as the vertical canals of the same ear gave normal responses, the vertical canals fibres are not in the inferior cerebellar peduncle. This patient has been examined at intervals for over two years and on each occasion invariably shows the same clean-cut phenomena.

The following cases would seem to suggest that the horizontal canal fibres are externally located in the brain-stem and that the vertical canals fibres are mesially located. One case showed a complete deafness in the left ear and no responses whatever from either the horizontal canal or from the vertical canals. The right ear showed normal hearing, normal responses from the right horizontal canal, but no responses whatever from the right vertical canals. Autopsy showed tumor of left cerebellar hemisphere, in the biventral lobe, involving the VIII Nerve in the left cerebello-pontile angle; the tumor was pressing against the brain-stem. This suggests that the pressure on the left side was sufficient to involve the right vertical canals fibres, which would therefore seem to be nearer the median line, and that the horizontal canal fibres of the right side which were not involved by this pressure would seem to be more externally located, beyond the influence of the pressure. A similar case (Mrs. R., page 412) showed complete deafness of the left ear and an absence of responses from the left horizontal and the left vertical canals. On the right side there were no responses from the vertical canals, whereas the horizontal canal gave normal reactions. It will be noted that these phenomena are identical with those appearing in the case just quoted. A decompression operation was done by Dr. J. Chalmers Da Costa; one week later another examination showed that not only the horizontal canal fibres of the right side reacted normally, but also the vertical canals fibres of the right side. This rather strikingly suggests that the pressure on the right vertical canals fibres had been relieved by the decompression.

That the fibres conveying impulses to the eyes, resulting in nystagmus, are distinct and separate from those conveying impulses to the cerebral cortex, producing vertigo, is borne out by the following cases: (1) Twenty-four cases of spontaneous nystagmus, unassociated with any vertigo whatever; (2) innumerable cases of spontaneous vertigo, unassociated at any time with nystagmus; (3) six patients who after ear-stimulation showed a normal nystagmus both in amplitude and duration, but no vertigo; (4) a patient who after ear-stimulation had no nystagmus whatever and yet experienced normal vertigo. The normal vertigo was proven by the exhibition of normal past-pointing and falling.

That the fibres conveying the impulses producing vertigo go through the cerebellum en route to the cerebrum, is made highly probable in that without exception all of our cases in which operation or autopsy has demonstrated a cerebellar lesion, have shown vertigo to be markedly subnormal or absent after ear-stimulation.

Summarizing, we are confident that the fibres from the horizontal canal and the fibres from the vertical canals have separate pathways in the brain-stem. This is absolute. That the horizontal canal fibres are confined to the medulla oblongata and enter the cerebellum through the inferior cerebellar peduncle, and that the vertical canals fibres ascend into the pons and enter the cerebellum through the middle peduncle, we believe to be highly probable; but we feel that the evidence to date is not sufficiently large to make absolute the *exact* course of the fibres. Therefore the tracts that we are about to present are of course not to be regarded as final. They do, however, represent the logical conclusions from our evidence to date. Furthermore, when used as a working basis for the examination of patients, this conception of the vestibular pathways has furnished diagnostic data which have proven surprisingly reliable. Further observation by ourselves and others may no doubt modify or change our conception of the exact course of the fibres. In the main essentials, however, these tracts may be regarded as accurate and in presenting them we will speak *dogmatically* for the sake of clearness.

## Tracts in Detail

From the various end-organs within the labyrinth, namely, the Corti's organ of the cochlea, the maculæ of the utricle and saccule, and the cristæ of the three semicircular canals, nerve-fibres enter the internal auditory meatus, where they all unite in one common bundle to form the VIII Nerve. At the point of entrance of the VIII Nerve into the medulla oblongata, the nerve-fibres again divide into two main trunks, namely, the auditory and vestibular portions. It is generally recognized that the auditory portion in turn divides into two separate paths, an anterior and a posterior.

Our observation which so far as we know is new, is that the *vestibular* branch in a similar way divides into two distinct paths. One path consists of the fibres from the horizontal canal; the other path consists of the fibres from the vertical canals (Fig. 32). Summarizing then, the VIII Nerve at its entrance to the medulla oblongata divides as follows: (1) Auditory, which divides into (*a*) anterior tract, and (*b*) posterior tract. (2) Vestibular, which divides into (*a*) horizontal canal fibres, and (*b*) vertical canals fibres.

The VIII Nerve may be likened to a string which is frayed—separated into its constituent strands—at both ends. The different fibres which go to make up the nerve, at one end are connected with the various end-organs within the labyrinth, and at the other end again separate to pursue their different courses.

## Auditory Fibres

The auditory fibres begin in the Corti's organs of the cochlea, relay in the spiral ganglia in the cochlea and enter the VIII Nerve. At the entrance of the VIII Nerve into the medulla oblongata, an anterior branch runs mesially to the trapezoid body of the same and opposite side, and a posterior branch continues around the posterior border of the medulla oblongata, passing around the inferior cerebellar peduncle and becoming the so-called acoustic striæ in the floor of the IV ventricle. The fibres from the acoustic striæ then continue *forward* until they also enter the trapezoid body of the same and opposite side. From the trapezoid body the fibres continue to the posterior corpora quadragemina and the median geniculate body, from which they pass to the subthalamic region and continue by association fibres to the temporal lobe, in the posterior portion of the first and possibly second convolutions. It is most probable that *most* of the auditory fibres from one ear go to the temporal lobe of the opposite side; some of the fibres, however, go to the temporal lobe of the same side.

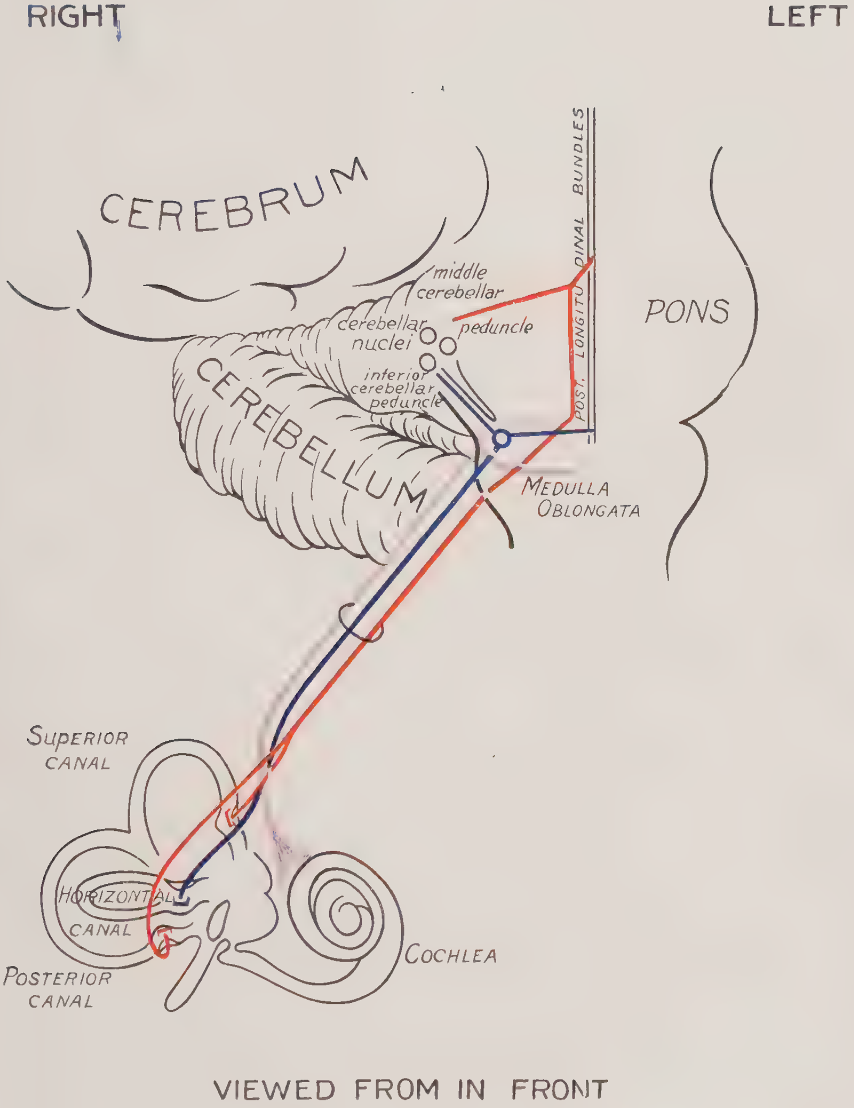


FIG. 32.—Pathways of fibres from cochlea, horizontal canal and vertical canals.

## VESTIBULAR FIBRES

After entering the medulla oblongata, the vestibular portion of the VIII Nerve divides into two tracts, the fibres from the horizontal canal and the fibres from the vertical canals.

We now consider the individual tracts. Each tract is best represented by a "Y" (Fig. 33). One arm of the "Y" eventually goes to the eye-muscles; the other arm goes to the cerebellum. The tract to the eye-muscles constitutes the vestibulo-ocular tract; impulses along this tract result in nystagmus. The tract to the cerebellum is the vestibulo-cerebellar tract; impulses along this tract and its further continuation to the *cerebral* cortex produce *vertigo.*

The horizontal canal fibres (Fig. 34) begin in the crista of the horizontal canal, relay in Scarpa's ganglion and enter the bundle of the VIII Nerve in which they enter the medulla oblongata. These fibres then pass through the medulla oblongata to the posterior external portion, at which point they enter Dieters' nucleus. It is at this point that the tract divides into two paths. One path goes through the nucleus triangularis toward the median line, entering the posterior longitudinal bundle, which is situated in the median line along the posterior aspect of the medulla oblongata and pons; the other path extends from Deiters' nucleus to the juxtarestiform body, which is the mesial portion of the inferior cerebellar peduncle, to the three vestibular nuclei within the cerebellum. These are the nucleus globosus, the nucleus emboliformis and the nucleus fastigii.

Consulting the diagrams, it will be observed that this tract is represented by a "Y" and after a *series* of diagrams of the same "Y," each diagram containing one more feature, the final diagram shows that the fibres on the one hand reach the posterior longitudinal bundle, which in turn connects it with the nuclei of the ocular muscles, and on the other hand the fibres reach the cerebellar nuclei.

Similarly, in the cristæ of the vertical canals—the superior and posterior—fibres originate which enter the VIII Nerve. At the entrance of the VIII Nerve into the medulla oblongata, however,

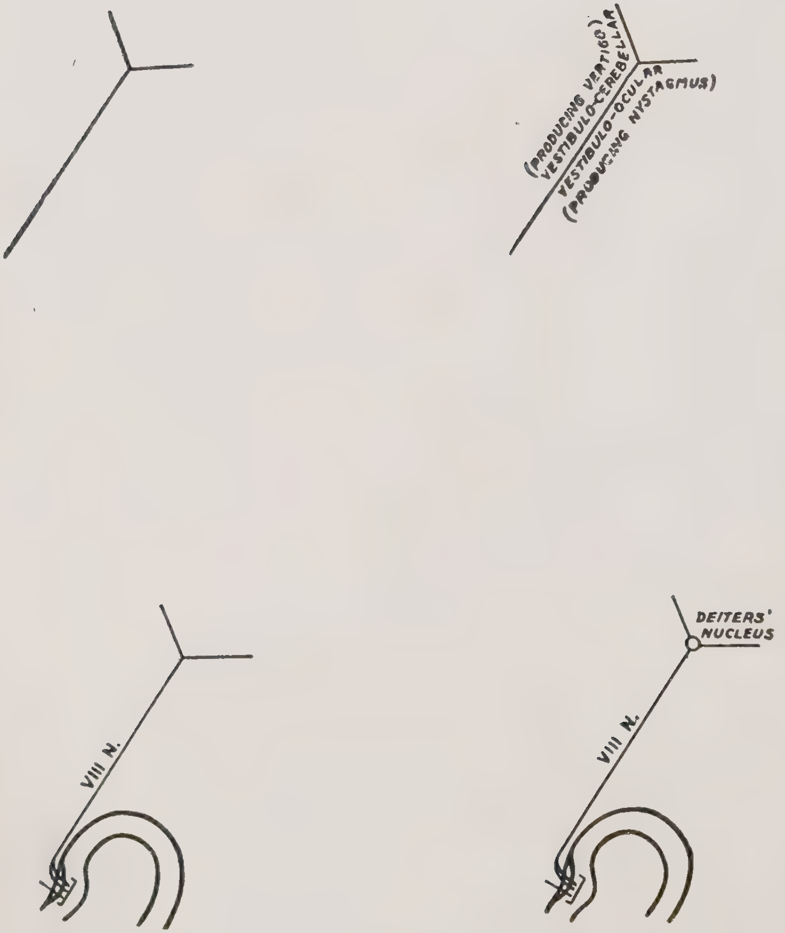


FIG. 33.—Consecutive series of diagrams; pathways of fibres from the right horizontal canal.

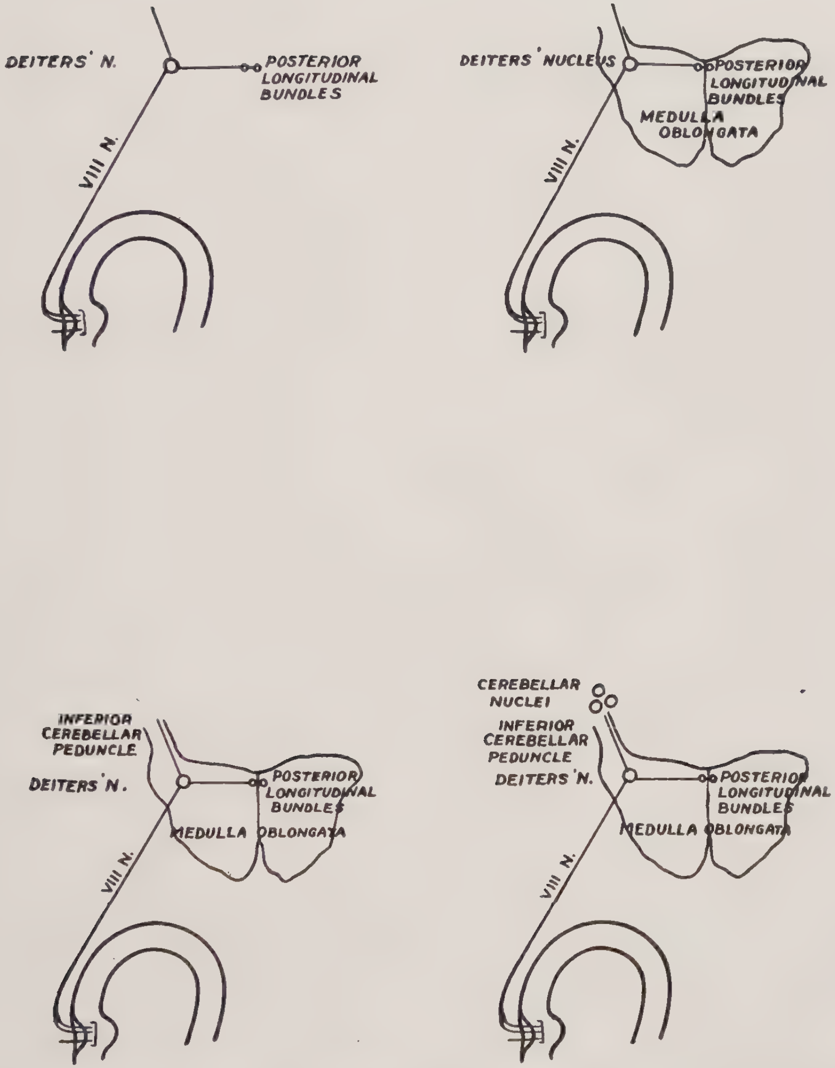

DEITERS' N.
POSTERIOR LONGITUDINAL BUNDLES
VIII N.
DEITERS' NUCLEUS
POSTERIOR LONGITUDINAL BUNDLES
MEDULLA OBLONGATA
VIII N.
INFERIOR CEREBELLAR PEDUNCLE
DEITERS' N.
POSTERIOR LONGITUDINAL BUNDLES
MEDULLA OBLONGATA
VIII N.
CEREBELLAR NUCLEI
INFERIOR CEREBELLAR PEDUNCLE
DEITERS' N.
POSTERIOR LONGITUDINAL BUNDLES
MEDULLA OBLONGATA
VIII N.

FIG. 34.—Continuation of consecutive series of diagrams; pathways of fibres from the right horizontal canal.

instead of going directly backwards to Deiters' nucleus, these fibres extend *upward* along the brain-stem in a course external to the posterior longitudinal bundle. At a point in the upper half of the pons, the exact location of which is not yet determined (an undetermined cell nest) there is also a branching into two arms of the "Y," similar to the branching of the horizontal canal fibres which occurs at Deiters' nucleus.

One arm of this "Y" enters the posterior longitudinal bundle, thus completing the afferent portion of the vestibulo-ocular tract of the vertical canals fibres. Impulses along this tract result in nystagmus. The other arm of the "Y" directly enters the cerebellum through the *middle* cerebellar peduncle and is distributed to the three cerebellar nuclei—globosus, emboliformis and fastigii. Impulses along this tract and its further continuation to the cerebral cortex produce vertigo.

Consulting the diagrams (Figs. 35 and 36), we notice that this tract is again represented by a "Y," and after a series of diagrams of the same "Y," each diagram containing an additional feature, the final diagram shows that the fibres on the one hand reach the posterior longitudinal bundle, and on the other hand the cerebellar nuclei. It will be noted that these diagrams of the vertical canals tracts are represented as a *longitudinal* section of the pons and medulla oblongata. This emphasizes the point that these fibres proceed *upward* in the brain-stem before dividing into their two separate paths.

If a cross-section were made of the upper portion of the medulla oblongata, just before it joins the pons, it would be shown, as in the accompanying diagram (Fig. 37), that the horizontal canal fibres are externally placed, and that the vertical canals fibres are mesially located, yet external to the posterior longitudinal bundle. Now if a section is made higher up, in the pons itself, it will be noted, as shown in the accompanying diagram (Fig. 37), that the vertical canals fibres alone are represented and that there are no horizontal canal fibres.

It will be noted that these vestibular tracts are carried on the one hand only to the posterior longitudinal bundle, and on the

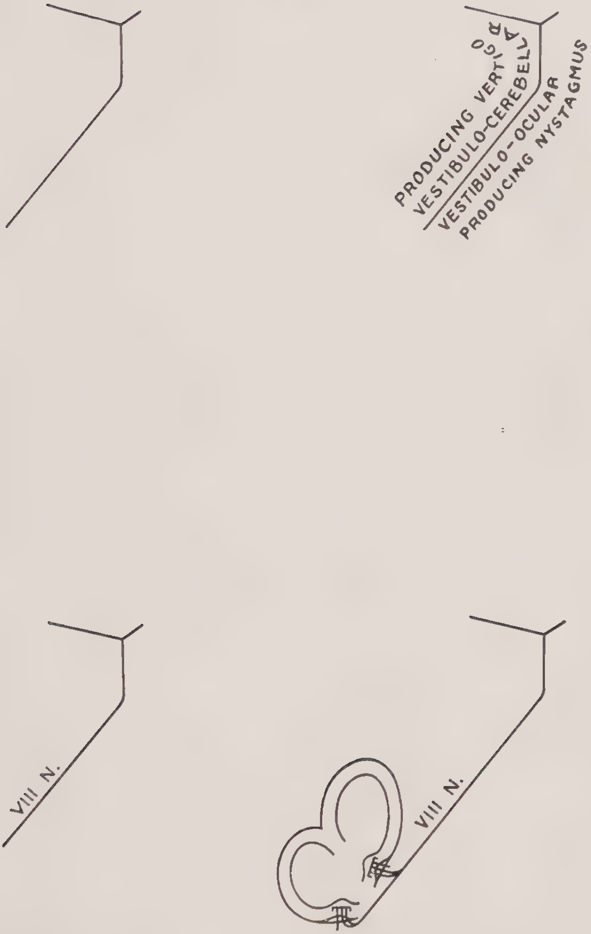


Fig. 35.—Consecutive series of diagrams; pathways of fibres from the right vertical canals.

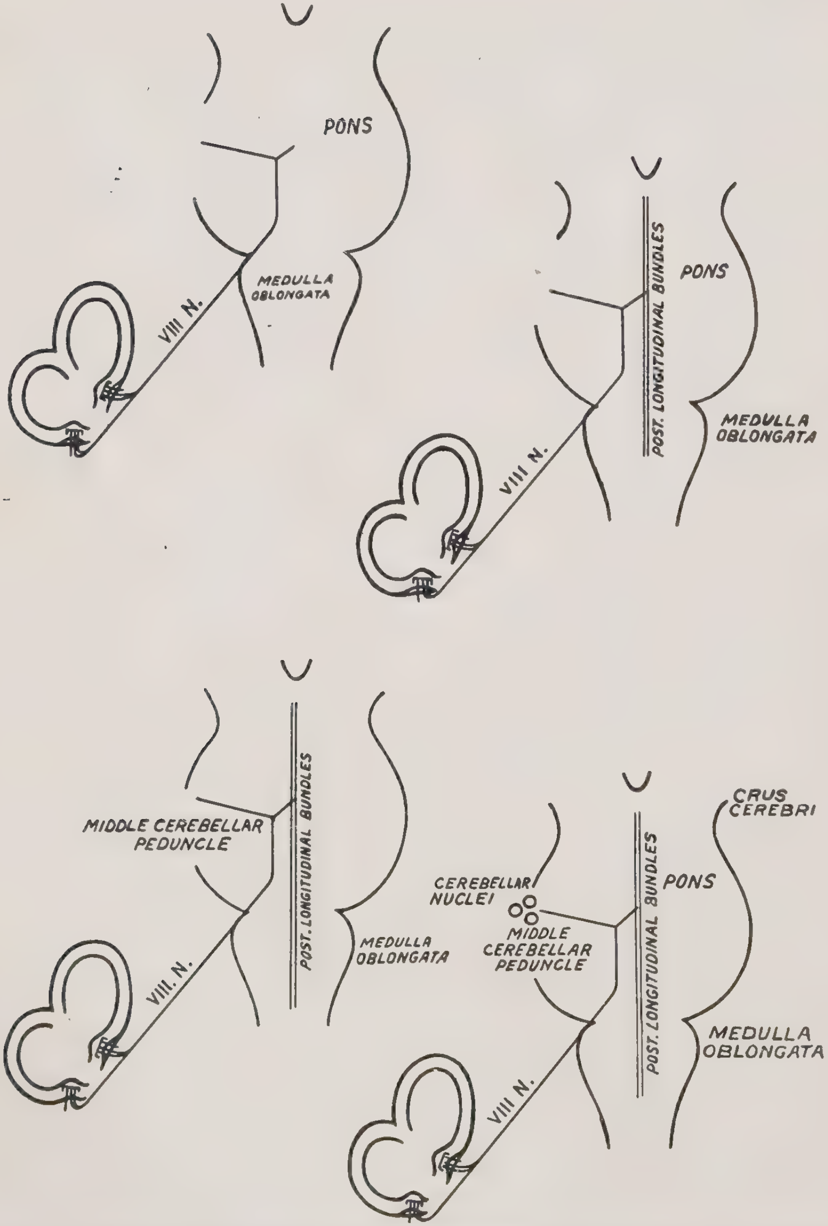


Fig. 36.—Continuation of consecutive series of diagrams; pathways of fibres from the right vertical canals.

other hand only to the cerebellar nuclei. Under subsequent chapters the paths will be carried further. Under the chapter on nystagmus the tracts will be continued from the posterior longitudinal bundle to the eye-muscles. In a similar way, under the chapter on vertigo, the tracts will be continued from the cerebellar nuclei through to the cerebral cortex.

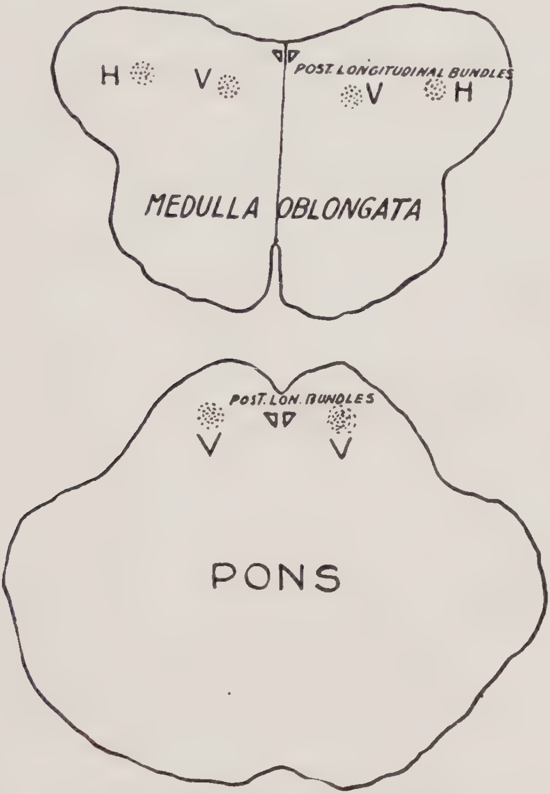


Fig. 37.—Diagram of cross-section at different levels of the brain-stem. 'H"=Horizontal canal fibres. "V"=Vertical canals fibres. In the pons there are no horizontal canal fibres.

# CHAPTER XV

## VESTIBULAR NYSTAGMUS

By the term of nystagmus in general is meant a twitching or to-and-fro movement of the eyes (Fig. 38). We will consider only that type of nystagmus which is produced by stimulation of the ear through the vestibulo-ocular tracts, and which is generally spoken of as vestibular nystagmus. Vestibular nystagmus is essentially a *rhythmic* movement of the eyes, in that it consists of a slow movement of the eyes in one direction, followed by a quick return movement in the opposite direction. The slow movement is generally spoken of as the slow *component* of nystagmus; it is the movement produced by the ear stimulation. The recovery, or quick movement in the opposite direction, is entirely cerebral in origin. It is this quick return movement of the eyes, however, that is the most conspicuous, and therefore the nystagmus is named, perhaps unfortunately, after this quick movement. We say that the nystagmus is to the left when the quick movement of the eyes is to the left. For example, if by ear stimulation the eyes are made to move toward the right, the quick return movement is to the left, and the nystagmus is spoken of as to the left. But it must be borne in mind that the real "ear-pull" in this case is to the right. In other words, the eye muscles receive impulses from two sources, from the ear for the slow component and from the cerebrum for the quick component. We will consider first the paths along which the impulses travel from the ear to the eye muscles. These tracts are (1) Horizontal semicircular canal tracts, (*a*) stimulus away from the ampulla, (*b*) stimulus toward the ampulla. (2) Vertical semicircular canals tracts, (*a*) stimulus away from the ampullæ, (*b*) stimulus toward the ampullæ.

These tracts, the horizontal canal tracts and the vertical canals tracts, produce the true vestibular slow movement of the eyes—the ear-pull.

In the preceding chapter it will be remembered that we have

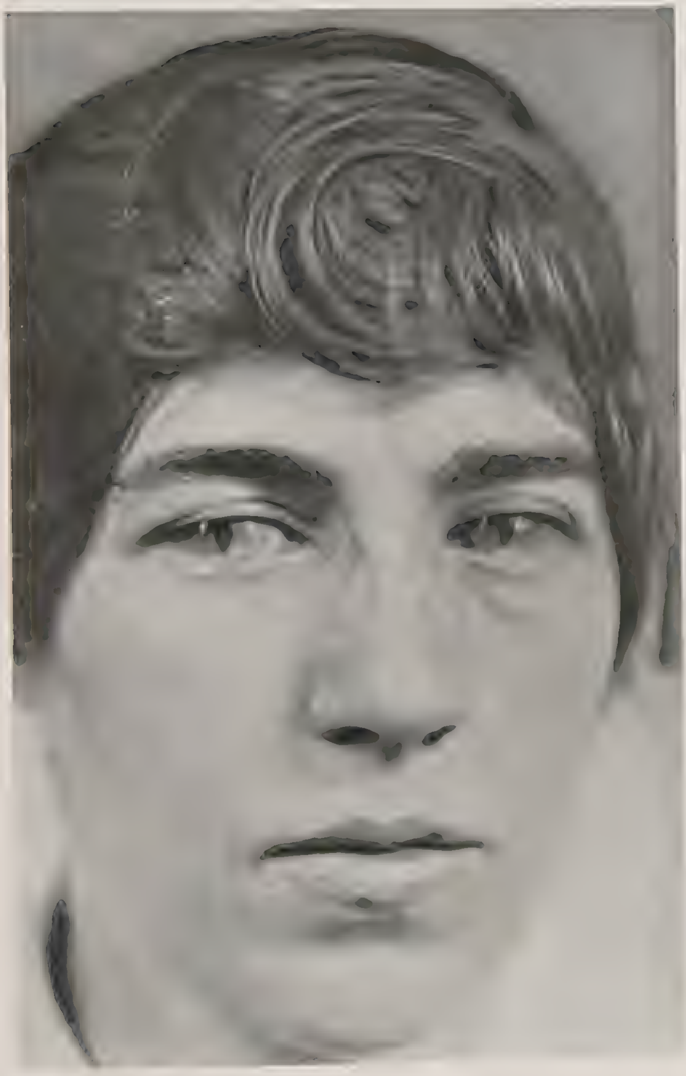


Fig. 38.—Horizontal nystagmus.

shown the paths from the ear up to the posterior longitudinal bundle. It is this bundle which connects the paths from the ear with the nuclei controlling the eye-movement. In addition the posterior longitudinal bundle makes possible the associated movement of the eyes.

(1) Horizontal canal tracts (Figs. 39 and 40). From the horizontal canal there are two separate tracts, one conveying the impulse when the current is away from the ampulla, and the other when the current is toward the ampulla. Both these tracts connect the horizontal canal with the III and VI ocular nuclei, and with these nuclei only and in fact only with that part of the III nucleus that controls the internal rectus muscle.

(*a*) Current away from the ampulla in the right horizontal canal. From the hair-cells influenced by this current, the fibres go in the VIII Nerve, through the medulla oblongata and Deiters' nucleus, and thence to the posterior longitudinal bundle. In the bundle these fibres continue to the VI nucleus of the right side and the III nucleus of the left side. The VI nucleus through the VI Nerve causes a contraction of the external rectus muscle of the right eye. The III nucleus through the III Nerve produces contraction of the internal rectus muscle of the left eye. The resultant action is the drawing of both eyes to the right.

Fig. 39 shows the current away from the ampulla in the horizontal canal; a single hair is put upon the stretch and the fibre from this hair proceeds through the VIII Nerve to Deiters' nucleus, and to the posterior longitudinal bundle through which it is distributed to the VI nucleus and nerve of the right side and to the III nucleus and nerve of the left side causing both eyes to be drawn to the right. The diagram is constructed in an attempt to show the actual relation in the head. A cross-section of the skull is being viewed from above. It will be noted that the eyes are drawn in the direction of the endolymph movement as expressed by the arrow in the semicircular canal.

(*b*) Current toward the ampulla in the right horizontal canal. From the hair-cells influenced by this movement the fibres continue in the VIII Nerve, through the medulla oblongata and Deiters' nucleus and thence to the posterior longitudinal bundle. In the

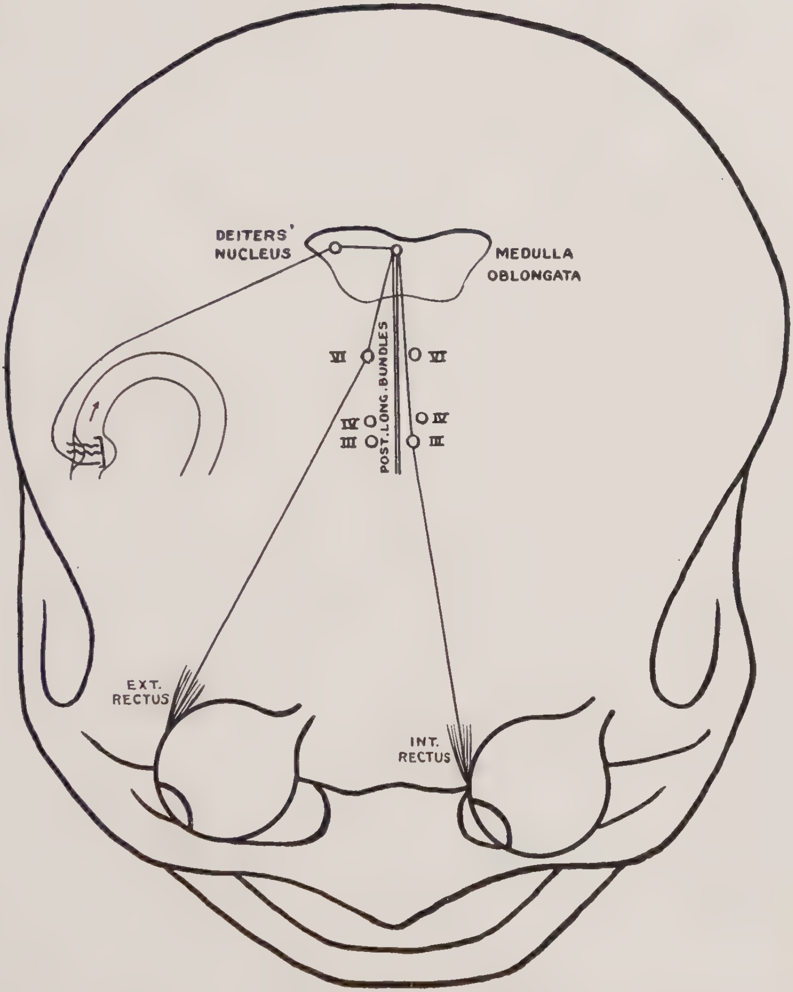


Fig. 39.—Vestibulo-ocular tract (producing nystagmus) of horizontal canal fibres (current away from ampulla). Eyes drawn in direction of endolymph movement. Horizontal section of head.

bundle these fibres continue to the III nucleus of the right side and to the VI nucleus of the left. The III nucleus of the right side through the III Nerve causes contraction of the internal rectus of the right eye. The VI nucleus through the VI Nerve causes contraction of the external rectus of the left eye. The resultant action is the drawing of both eyes to the left.

Fig. 40 shows the current toward the ampulla in the right horizontal canal; the two hairs are put upon the stretch and the fibres from these two hairs continue through Deiters' nucleus and the posterior longitudinal bundle to the right III nucleus and nerve and to the left VI nucleus and nerve, causing the eyes to be drawn to the left. It will be noted that the eyes are drawn in the direction of the endolymph movement as shown by the arrow in the semicircular canal.

It will be observed in passing that in both these reactions we have produced only a *conjugate deviation* of the eyes, in the one case to the right and in the other case to the left. *This is not nystagmus.* This is only the slow movement of nystagmus; the quick return movement of the eyes in the opposite direction which would complete the nystagmus will be considered later.

(*a*) Current away from the ampullæ of the superior canal and of the posterior canal of the right ear. From the hair-cells influenced by this movement the fibres go through the VIII Nerve, ascend in the pons and are distributed by the posterior longitudinal bundles to the IV nucleus of the right side and the III nucleus of the left side. The IV nucleus of the right side through the IV Nerve produces contraction of the superior oblique muscle of the right eye, causing the right eye to rotate to the left. The III nucleus of the left side through the III Nerve produces contraction of the inferior oblique muscle of the left eye, causing the left eye to rotate to the left. The resultant action, therefore, is a rotary movement of both eyes to the left.

Fig. 41 shows the current away from the ampullæ of both the superior canal and the posterior canal. This current puts on the stretch the two hairs in the superior canal and in the posterior canal. The fibres from these four hairs pass through the VIII Nerve and in the upper half of the pons enter the posterior longi-

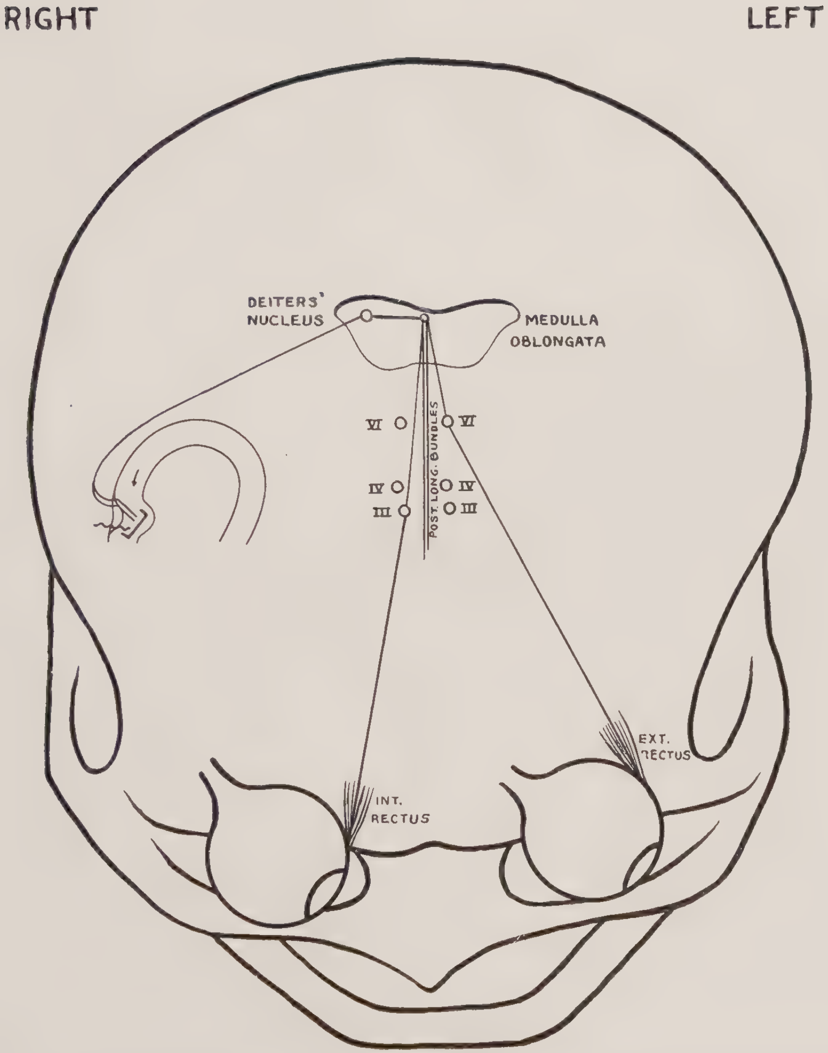
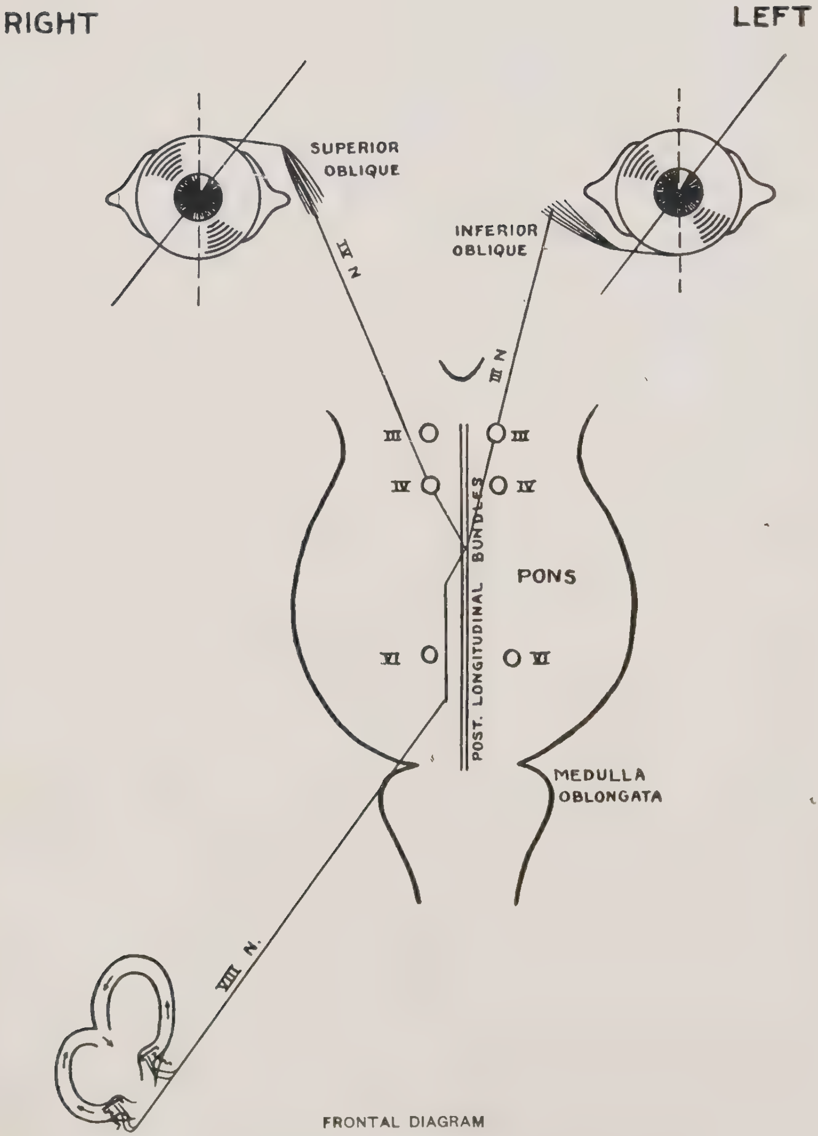


Fig. 40.—Vestibulo-ocular tract (producing nystagmus) of horizontal canal fibres (current away from ampulla). Eyes drawn in direction of endolymph movement.



FRONTAL DIAGRAM

VESTIBULO-OCULAR TRACT OF VERTICAL CANALS FIBRES (PRODUCING NYSTAGMUS) (CURRENT AWAY FROM AMPULLAE)

Fig. 41.—Eyes drawn in direction of endolymph movement

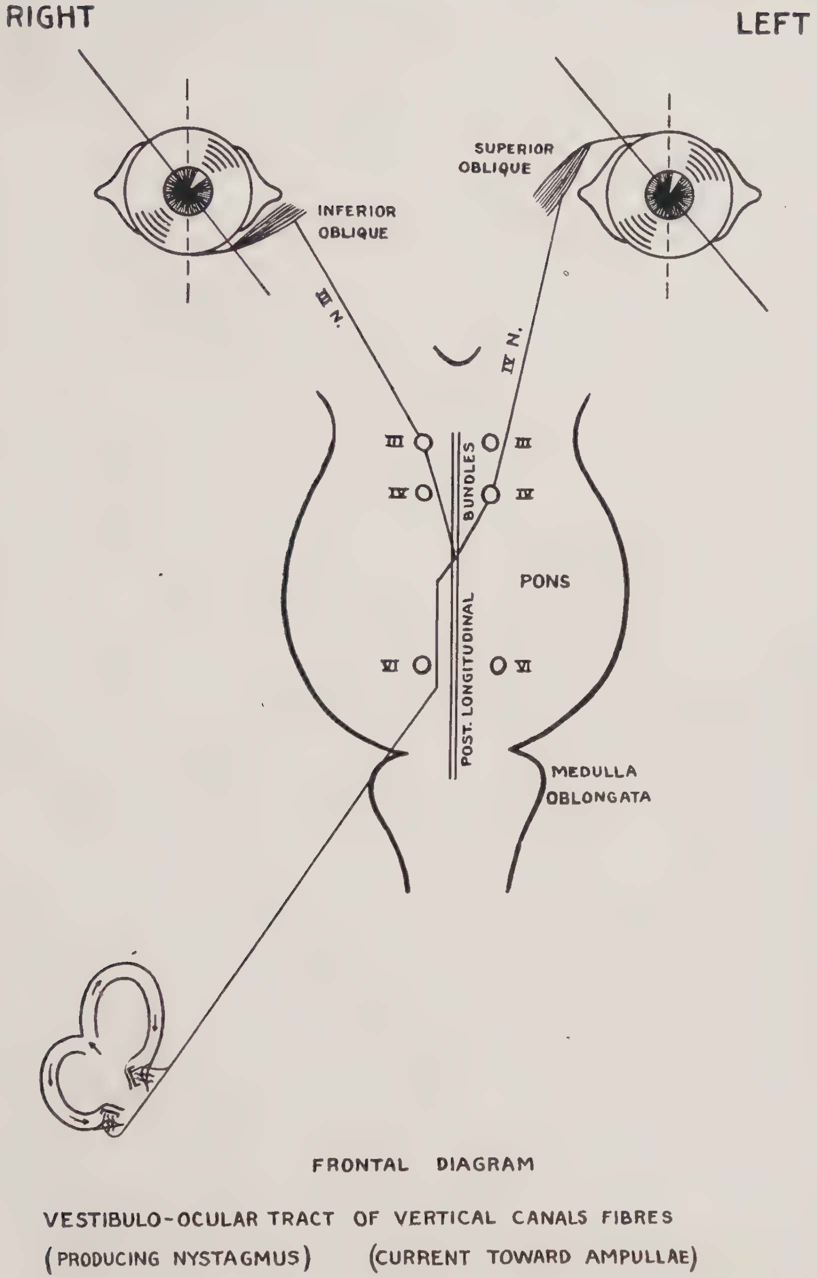


Fig. 42.—Eyes drawn in direction of endolymph movement.

tudinal bundles to be distributed to the IV nucleus of the right side and to the III nucleus of the left, causing the upper meridian of both eyes to be drawn to the left.

(*b*) Current toward the ampullæ of the vertical canals of the right ear, produces a stimulation of the III nucleus of the right side and the IV nucleus of the left side. The III nucleus of the right side through the III Nerve produces contraction of the inferior oblique muscle of the right eye, causing the right eye to rotate to the right. The IV nucleus of the left side through the IV Nerve produces contraction of the superior oblique muscle of the left eye, causing the left eye to rotate to the right. The resultant action, therefore, is the rotary movement of both eyes to the right.

The current toward the ampullæ (Fig. 42) puts on the stretch the one hair in the superior canal and the one hair in the posterior canal. Fibres from these two hairs are distributed to the III nucleus and nerve of the right side and to the IV nucleus and nerve of the left, causing the upper meridian of both eyes to be drawn to the right.

It must be mentioned in passing that the action of the oblique muscles is not a pure rotary movement. The superior oblique also causes the cornea to turn outward and downward. The inferior oblique causes the cornea to turn outward and upward. In the mechanism of a rotary movement of the eyeball, therefore, there is a definite action also of the superior *rectus* and of the inferior *rectus*. But as the main action is that of the oblique it is best for purposes of simplicity to consider them only.

Again it will be remembered that in both of these reactions from the vertical canals, we have produced only a *conjugate deviation of the eyes*—a rotary movement in the one case to the left and in the other case to the right. This is not nystagmus; this is only the slow movement of nystagmus. The quick return movement of the eyes in the opposite direction which would complete the nystagmus will now be considered.

## THE CEREBRAL FIBRES TO THE EYE-MUSCLE NUCLEI

The tracts already described have caused a slow conjugate deviation of the eyes. The horizontal canal causes a horizontal movement of the eyes to one side. The vertical canals cause a rotary movement of the eyes in one direction. This is not nystagmus. The cerebrum, conscious of the contraction of certain eye-muscles and the relaxation of others, recognizes that the eyes are drawn away from the normal position and sends impulses to the exactly opposite set of eye-muscle nuclei, causing the quick return. It is this slow drawing of the eyes out of the usual position and the quick return movement that together constitute nystagmus.

Example: the current away from the ampulla of the right horizontal canal has caused a conjugate deviation of the eyes to the right, by stimulation of the VI nucleus of the right side and the III nucleus of the left. The cerebral fibres then cause a stimulation of the III nucleus of the right side and of the VI nucleus of the left, causing the eyes to be jerked quickly to the left. This constitutes nystagmus to the left.

If an individual is under the influence of ether and his ears are douched, there is produced a conjugate deviation only. The cerebral impulses are wanting and there is no return movement.

The cerebrum is not conscious of the altered position of the eyes by the sense of sight; blind people, on ear examination, have an unusually *large* nystagmus. We do know that when the eyes are drawn out of the usual position, some eye muscles are contracted and others are relaxed; therefore, the best explanation is that stimuli from these muscles to the cerebral cortex notify the higher centres of the altered position of the eyes.

The cerebral centre for the quick component of nystagmus has not been definitely located. The two paths travel down through the internal capsules into the uppermost portion of the posterior longitudinal bundles at the point where the crura cerebri unite to form the pons. The fibres are then distributed by means of the posterior longitudinal bundles to the various eye-muscle nuclei. The quick component to the left of both eyes is probably controlled by a centre in the right cerebral hemisphere, and the left cerebral hemisphere probably controls the quick component of both eyes to the right. This is strongly suggested by data in case, with necropsy, given in detail on page 382, case 21.

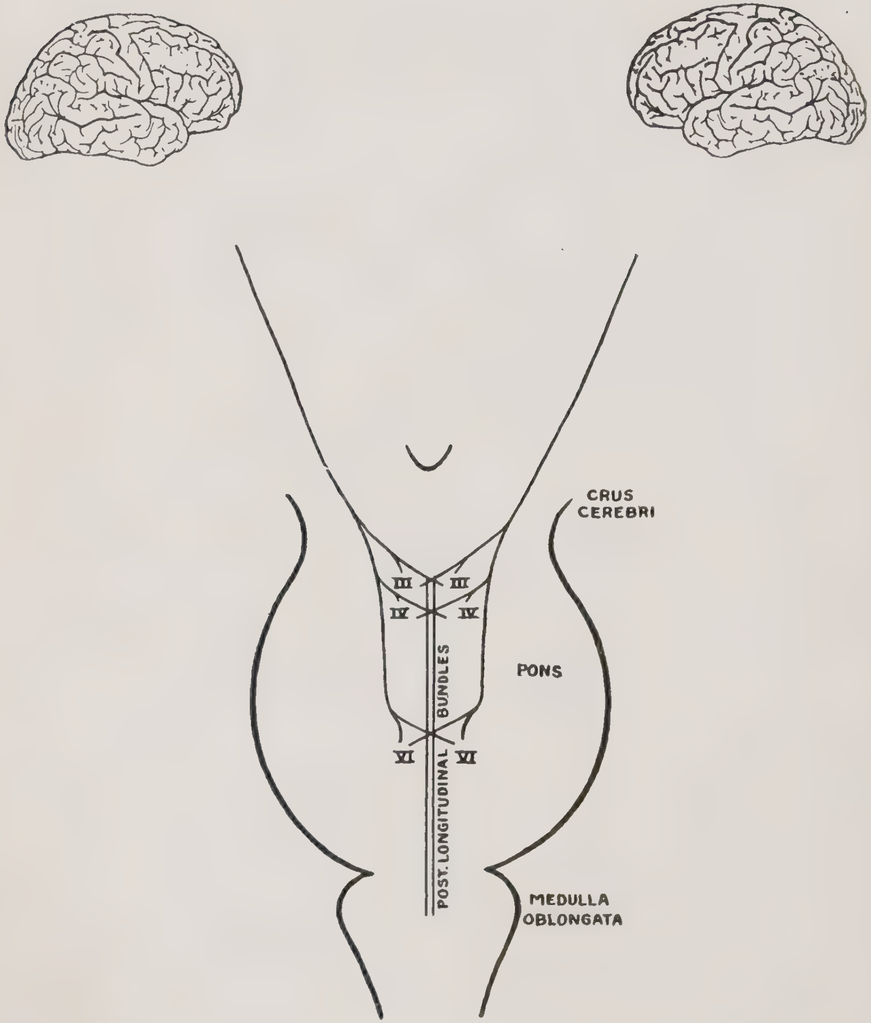


Fig. 43.—Cerebral fibres to eye-muscle nuclei (the quick component of nystagmus).

### Planes of Nystagmus

There are three main planes of vestibular nystagmus—horizontal, frontal and sagittal. Nystagmus in a horizontal plane consists of a movement of the eyes toward the right or toward the left. Nystagmus in the frontal plane consists of a rotary movement of the eyes on their anteroposterior axes, either toward the right or toward the left. Nystagmus in the sagittal plane consists of a vertical movement of the eyes either upward or downward. There is an exact and constant relation between the planes of the semicircular canals and the planes of the nystagmus produced by ear stimulation. The horizontal canal, as its name implies, is approximately in a horizontal plane. Stimulation, therefore, of only the horizontal canal produces a pure horizontal nystagmus. The fibres from the horizontal canal are distributed exclusively to the external and internal recti muscles. This relation is definitely established and is very suggestive of itself that there must be different and entirely separate tracts from the different canals. The best explanation is that the horizontal canal, for example, has its own fibres which are *insulated* from all the other fibres throughout its entire course, until the stimulus reaches those particular eye muscles with which this semicircular canal is connected, namely the external and internal recti.

The production of a horizontal nystagmus by stimulation of the horizontal canal is relatively simple to understand. The great difficulty has been in establishing the relation of the *vertical* canals to the plane of the nystagmus which they produce. Perhaps the simplest explanation is as follows: The superior canal lies in a plane halfway between the frontal and the sagittal, and viewed from the outside of the head, it runs from a forward position, backward. The posterior canal likewise lies in a plane halfway between the frontal and the sagittal, only it runs forward. The superior canal, for instance, is not placed in the frontal plane, nor is the posterior canal placed in the sagittal plane; the essential feature is that these two vertical canals *act together*. We know this for the following reasons: (1) They are both in the vertical axis so that any movement in a vertical axis influences both. Experimentally, either douching with the head upright or turning with the head forward or backward, affects both of these canals. (2) Anatomically at their innermost point, they unite in a *common crus*. The movement, therefore, of the endolymph in one canal cannot take place without a corresponding movement of the endolymph in the other.

The plane of the resulting nystagmus then is the *resultant of the planes of the vertical canals*. This was first brought out by E. R. Lewis. With the head 120° forward, on turning, the plane of the movement of the head is frontal.

This frontal plane exactly bisects the planes of the two vertical canals. It will be noted that in this position the superior canal has an arc of 45° inferior to this frontal plane; and that the posterior canal has an arc of 45° superior to the frontal plane. It is the contribution, therefore, from these two canals that produces a reaction in the frontal plane. Clinically this is true—a rotary nystagmus is produced in the frontal plane. In a similar way, if the patient incline the head 90° toward the shoulder and is turned in that position, the plane of the turning is in the sagittal plane of the head. It will be observed that the superior canal is external to the sagittal plane by an arc of 45°, and that the posterior canal is also external to the sagittal plane by an arc of 45°. In other words, these two canals working together produce the combined result that one canal would produce if placed in the sagittal plane. Clinically this also is true—a nystagmus is produced in the sagittal plane, a vertical nystagmus upward or downward.

In brief, the horizontal canal produces a horizontal nystagmus. The vertical canals produce a vertical nystagmus in the frontal plane when the vertical canals are influenced in the frontal plane. The vertical canals produce a vertical nystagmus in the sagittal plane when the vertical canals are influenced in the sagittal plane.

## METHODS OF PRODUCING VESTIBULAR NYSTAGMUS

In order to produce a nystagmus in any plane it is merely necessary to produce a movement of the endolymph in the corresponding plane of the labyrinth. The two routine methods of producing movement of the endolymph within the labyrinth are (1) turning, and (2) douching.

(1) Turning. For this method, a person is placed in a smoothly revolving chair and turned. At the beginning of the turning the endolymph within the semicircular canal lags behind (Fig. $44^{a}$).

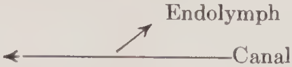


Fig. $44^{a}$.

After a few turns, however, usually ten, the endolymph has *caught up* with the movement of the canal and moves at the same rate of speed as the canal (Fig. $44^{b}$).

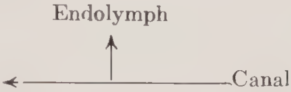


Fig. $44^{b}$.

The chair is then stopped. The momentum of the endolymph causes it to continue to move in the direction of turning (Fig. 45).

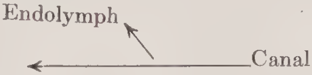


Fig. 45.

With the head in the upright position (or more accurately 30° forward, which puts the horizontal canals in exactly the horizontal plane), the horizontal canals of both sides are stimulated. In the right horizontal canal the endolymph current is away from the ampulla, and in the left horizontal canal the endolymph is toward the ampulla. The current away from the ampulla of the right side produces a movement of the eyes toward the *same side,* namely, the *right.* The current toward the ampulla of the left canal produces a pulling of the eyes to the opposite side, namely, the *right.* In other words, the two canals supplement each other in that they each produce a pulling of the eyes to the right. It has been proven that the stimulus toward the ampulla of the horizontal canal is twice as strong as the stimulus away from the ampulla. In this instance, therefore, two-thirds of the reaction is due to the left canal and one-third to the right canal. (Of course the *nystagmus* in this instance would be to the left. We will discuss only the vestibular or slow component.)

During the turning the canal is moving away from the endolymph (Fig. 38). The current, therefore, in the right canal is toward the ampulla; in the left canal it is away from the ampulla. The result, therefore, during the turning is a vestibular pull of the eyes to the left, that is to say, a *nystagmus* toward the right. It was this nystagmus *during* turning that was studied by the original investigators. A revolving platform was constructed on which the examiner was rotated with the patient, and with his finger placed over the patient's eyelids he could *feel* the twitching movement. The *after*-turning nystagmus, however, is so much more easily observed that the old method has become obsolete. It is only of academic interest to note that during the turning the canal moves away from the endolymph, and that after stopping the endolymph moves away from the canal. Also that the nystagmus after turning, for this reason, is in exactly the opposite direction to the nystagmus during the turning.

### Table of Nystagmus After Turning

For the convenience of the reader, we now present a tabulated list of the various forms of nystagmus produced after turning with the head in various positions.

Turning to the right with the head upright produces a horizontal nystagmus to the left. →

Turning to the left with the head upright produces a horizontal nystagmus to the right. ←

Turning to the right with the head 60° backward produces a rotary nystagmus to the left. ↷

Turning to the left with the head 60° backward produces a rotary nystagmus to the right. ↶

Turning to the right with the head 90° forward produces a rotary nystagmus to the left. ↷

Turning to the left with the head 90° forward produces a rotary nystagmus to the right. ↶

Turning to the right with the head inclined toward the right shoulder produces a vertical nystagmus downward. ↓

Turning to the left with the head inclined toward the right shoulder produces a vertical nystagmus upward. ↑

Turning to the right with the head inclined toward the left shoulder produces a vertical nystagmus upward. ↑

Turning to the left with the head inclined toward the left shoulder produces a vertical nystagmus downward. ↓

(2) Caloric. The caloric test of Barany consists in douching a person's ear with cold or hot water. Recalling that the essential feature in the production of nystagmus is a movement of the endolymph within the labyrinth, we accomplish it in this test by either cooling or warming one spot of the labyrinthine capsule. The endolymph at that point is either cooled or warmed and in consequence either sinks or rises. Cooling increases the specific gravity of the endolymph and causes it to sink. Heating decreases the specific gravity and causes the endolymph to rise. In both instances a current is established, but in the exactly opposite direction. This naturally takes place only in an up-and-down direction. The temperature of the endolymph is approximately 100°. In order to produce this reaction the water is used either 20° to 40° *below* the body temperature if we desire to cool the endolymph, or 10° to 15° *above* the temperature of the endolymph, if we desire to heat it.

With the head in the upright position, douching, of course, influences the vertical canals (see Fig. 2). This consequently produces a nystagmus in the frontal plane. Cold water causes a flow of the endolymph downward toward the ampullæ of the vertical canals, producing a vestibular pull of the upper meridian of the eyes toward the same side; in other words, a *nystagmus* to the opposite side. Hot water with the head upright, naturally produces exactly the opposite reaction, namely, as the flowing is away from the ampullæ, the vestibular pull of the eyes is to the opposite side and the nystagmus to the same side. With the head inclined 60° backward, the horizontal canal then assumes the vertical position. Douching in this position with cold water consequently causes a flow of the endolymph away from the ampulla, producing a vestibular pull of the eyes toward the same side in the horizontal plane of the head, that is to say, a nystagmus to the opposite side. This reaction being produced by the horizontal canal is, of course, a horizontal nystagmus. Similarly if the head is inclined 120° forward and the ear douched with cold water, the endolymph current is toward the ampulla. This produces a vestibular drawing of the eyes to the opposite side, that is to say, a nystagmus to the same side. As it was the horizontal canal that was affected there is produced a nystagmus in the horizontal plane.

For the convenience of the reader, the various reactions obtained by douching are tabulated below.

### Nystagmus After Douching

Douching the right ear with the head upright, water 68°, produces a rotary nystagmus to the left. ↷

Douching the left ear with the head upright, water 68°, produces a rotary nystagmus to the right. ↶

Douching the right ear with the head 60° backward, water 68°, produces a horizontal nystagmus to the left. →

Douching the left ear with the head 60° backward, water 68°, produces a horizontal nystagmus to the right. ←

Douching the right ear with the head 120° forward, water 68°, produces a horizontal nystagmus to the right. ←

Douching the left ear with the head 120° forward, water 68°, produces a horizontal nystagmus to the left. →

Douching the right ear with the head upright, water 112°, produces a rotary nystagmus to the right. ↶

Douching the left ear with the head upright, water 112°, produces a rotary nystagmus to the left. ↷

Douching the right ear with the head 60° backward, water 112°, produces a horizontal nystagmus to the right. ←

Douching the left ear with the head 60° backward, water 112°, produces a horizontal nystagmus to the left. →

Douching the right ear with the head 120° forward, water 112°, produces a horizontal nystagmus to the left. →

Douching the left ear with the head 120° forward, water 112°, produces a horizontal nystagmus to the right. ←

It must not be considered that all these tables of reactions must be remembered. After one has become familiar with the vestibular tests he comes to know what reactions to expect in the normal, just as he is familiar with the use of the tuning-fork or any other routine procedure. Further, it is not necessary to remember a single reaction. All that we need to remember is *the eyes are always drawn in the direction of the endolymph movement*. It is an unfailing rule and makes this apparently complex subject extremely simple.

# CHAPTER XVI

## VESTIBULAR VERTIGO

Vertigo from whatever cause is a subjective sensation of a disturbed relationship to objects in space. This disturbance is necessarily due to an alteration, either stimulating or depressing, of some portion of the *vestibular apparatus.* Inasmuch as the tests of the kinetic-static labyrinth disturb the vestibular apparatus, vertigo is necessarily a resulting phenomenon. It is distinctly a cerebral disturbance resulting from impulses carried from the ear to the cerebral cortex.

In Chapter XIV the tracts producing vertigo are described as extending from the ear, through the VIII Nerve, through the brainstem, and through the cerebellar peduncles as far as the cerebellar nuclei (Figs. 32, 33, 34, 35 and 36). There are two separate afferent paths from the ear to the cerebral cortex, resulting in vertigo from ear-stimulation: (1) The horizontal canal tracts, and (2) the vertical canals tracts.

Fig. 46 shows the current away from the ampulla in the right horizontal canal. A single hair is put upon the stretch; the nerve-fibre from the hair is represented as entering the VIII Nerve, going to Deiters' nucleus, entering the cerebellum through the inferior peduncle and going to the vestibular cerebellar nuclei. From the nuclei the heavy line represents the main pathway to the cerebral cortex, passing from the cerebellum through the right superior cerebellar peduncle in which it decussates in the base of the cerebral crura, and continuing through the special sensory tract to the posterior portion of the temporal lobe of the left side. Another pathway from the cerebellar nuclei is represented by a thinner line extending through the right superior peduncle, but instead of decussating to the opposite side, this minor path continues through the cerebral crus of the same side to the temporal lobe of the same side. Those pathways from the fibres influenced by a current away from the ampulla in the right horizontal canal produce a subjective sensation of turning to the *left.* It will be noted that the vertigo is in a direction opposite to the direction of the endolymph movement as represented by the arrow in the semicircular canal.

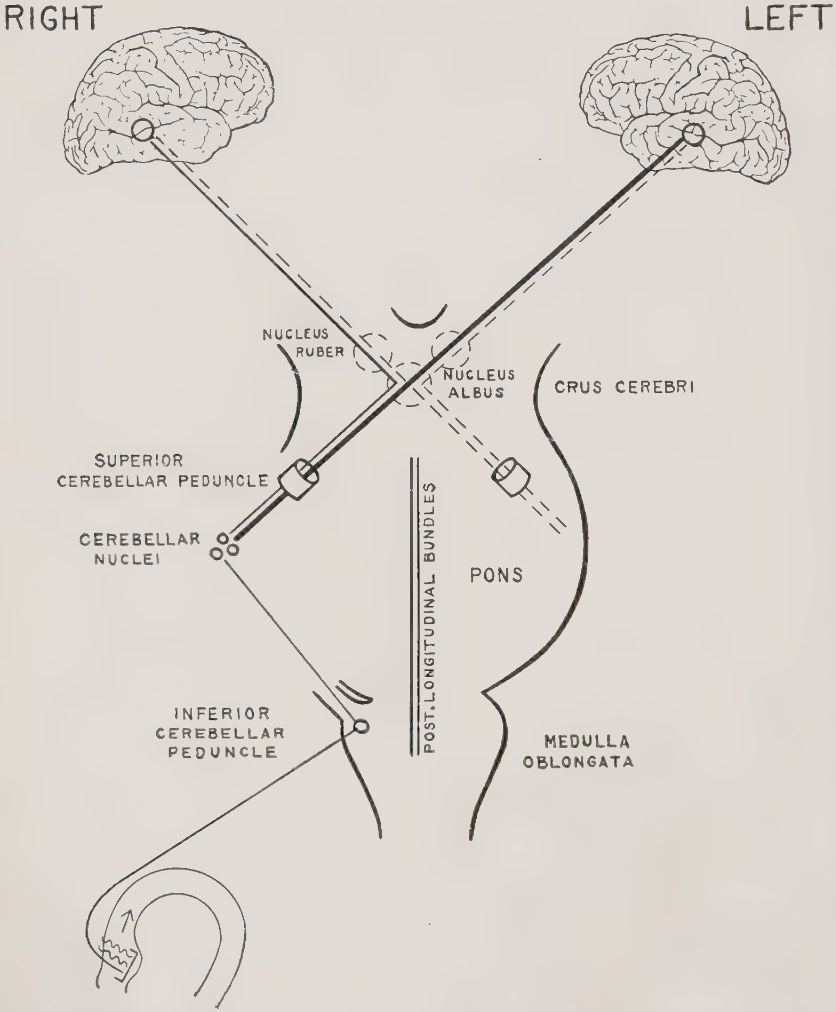


Fig. 46.—Vestibulo-cerebello-cerebral tract for horizontal canal fibres (producing vertigo) (current away from ampullæ).

Figure 47 is the same as the preceding diagram, except that the current toward the ampulla puts upon the stretch the *two* hairs on the other side of the crista. This stimulation causes a

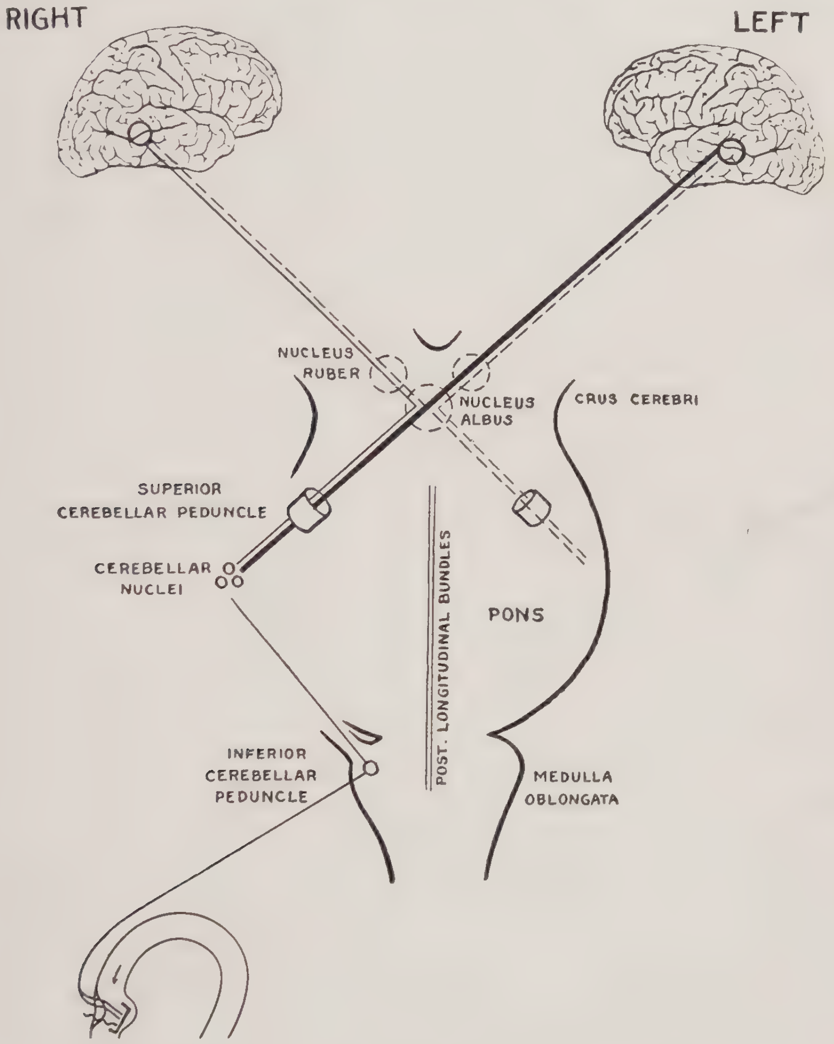


Fig. 47.—Vestibulo-cerebello-cerebral tract for horizontal canal fibres (producing vertigo).

subjective sensation of turning to the right—in a direction opposite to the endolymph movement.

Figure 48 shows the vertical canals with the current away from the ampullæ. The two hairs in the superior canal and the two hairs in the posterior canal are put upon the stretch. From these hairs the fibres pass through the VIII Nerve, ascend in the pons to a point above the middle of the pons, where they enter the cerebellum through the middle cerebellar peduncle and go to the cerebellar nuclei of the same side. From the cerebellar nuclei the course of these fibres is the same as that of the horizontal canal

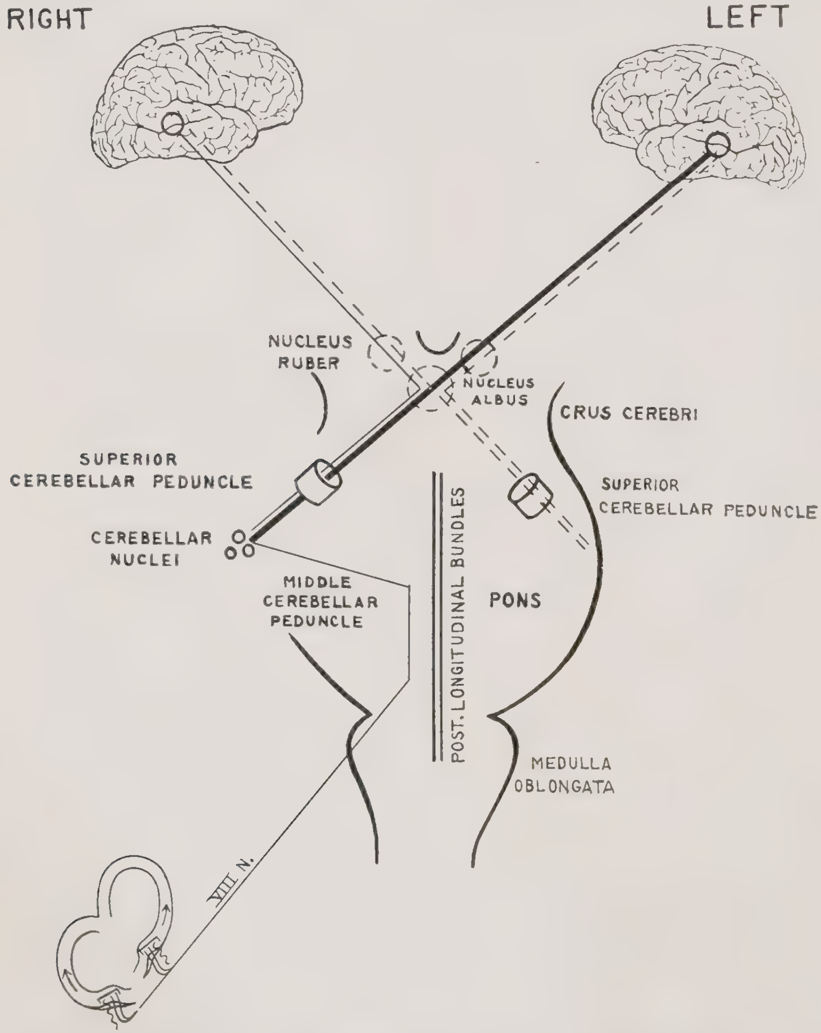

RIGHT
LEFT
NUCLEUS RUBER
NUCLEUS ALBUS
CRUS CEREBRI
SUPERIOR CEREBELLAR PEDUNCLE
SUPERIOR CEREBELLAR PEDUNCLE
CEREBELLAR NUCLEI
MIDDLE CEREBELLAR PEDUNCLE
POST. LONGITUDINAL BUNDLES
PONS
MEDULLA OBLONGATA
VIII N.

Fig. 48.—Vestibulo-cerebello-cerebral tract for vertical canals fibres (producing vertigo) (current away from ampullæ).

fibres. This stimulation, of the current away from the ampullæ, produces a subjective sensation of falling to the *right*—in a direction opposite to the endolymph movement.

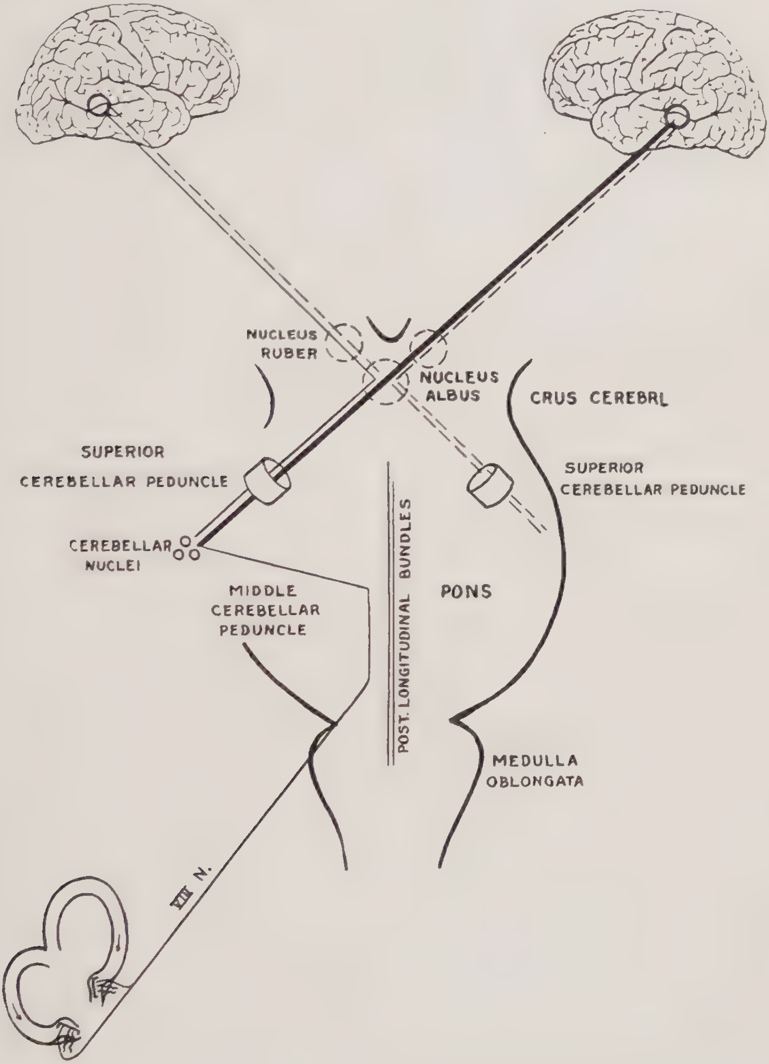

NUCLEUS RUBER
NUCLEUS ALBUS
CRUS CEREBRI
SUPERIOR CEREBELLAR PEDUNCLE
SUPERIOR CEREBELLAR PEDUNCLE
CEREBELLAR NUCLEI
POST. LONGITUDINAL BUNDLES
PONS
MIDDLE CEREBELLAR PEDUNCLE
MEDULLA OBLONGATA
VIII N.

Fig. 49.—Vestibulo-cerebello-cerebral tract for vertical canals fibres (producing vertigo).

Figure 49 is identical with the preceding diagram except that the current in the vertical canals is *toward* the ampullæ, putting upon the stretch the single hair in the superior canal and the single hair in the posterior canal. This stimulation causes a subjective sensation of falling to the *left*—in a direction opposite to the endolymph movement.

The cortical centre for the reception of vestibular impulses has been postulated by Mills to be in the posterior portion of the first and second temporal convolutions, adjacent to the cortical area for hearing. Mills considers that there is such a centre in each cerebral hemisphere and agrees with Dana that this centre is probably more highly developed in the right temporal region. It is not to be considered that the sensation of vertigo is appreciated in the two cortical areas in the two temporal lobes. These vestibular centres constitute the termini of the vestibular fibres; association fibres in turn connect these centres with other parts of the cerebral cortex, particularly the frontal lobes.

## THE PLANES OF VESTIBULAR VERTIGO

The vestibular reaction of vertigo, just like the reaction of nystagmus, manifests itself in certain definite planes: (1) Sensation of turning in the horizontal plane, either from the right to the left or from the left to the right. (2) Sensation of turning in the frontal plane, consisting of a sensation of *falling* to the right or falling to the left. (3) Sensation of turning in the sagittal plane, consisting of a sensation of *falling* forward or backward.

The sensation of movement in the horizontal plane is produced only by the horizontal canal or canals. This is illustrated by turning the patient with the head 30° forward.

The sensation of turning in the frontal plane is produced only when the vertical canals are influenced in the frontal plane. When the patient is turned with the head forward or backward, the plane of the rotation is frontal. Now if after turning (Fig. 50) the head is kept forward or backward, the subjective sensation is of turning in the frontal plane, which in this position of the head is *parallel to the floor.* The sensation is therefore the same as after turning with the head upright, namely, a movement about one's own axis either to the right or to the left. As it is a sensation of turning in a plane parallel to the floor, it is not unpleasant. If, however, after turning, the patient's head is raised to the upright position, the frontal plane then becomes at *right angles* to the floor and the sensation is that of falling in the frontal plane either to the right or to the left, and is therefore incidentally unpleasant.

The sensation of turning in the sagittal plane is produced only when the vertical canals are influenced in the sagittal plane. When the patient is turned with the head inclined well over toward the shoulder, the plane of the rotation is sagittal. Now if after turning, the head is kept in this same position toward the shoulder, the subjective sensation is of turning in the sagittal plane, which in this position of the head is *parallel to the floor.* The sensation is therefore the same as after turning with the head upright, namely a movement about one's own axis either to the right or to the left. As it is a sensation of turning in a plane parallel to the floor, it is not unpleasant. If, however, after turning, the patient's head is raised to the upright position, the sagittal plane then becomes at *right angles* to the floor and the sensation is that of falling in the sagittal plane; this consists of a feeling of pitching forward or backward, and is therefore incidentally unpleasant.

In speaking of this unpleasantness resulting from endolymph movement in the vertical plane, we may recall the illustration of seasickness. Barany calls attention to the fact that a ship at sea tosses in various *planes:*

(1) The horizontal plane, from right to left. This movement, however, is usually very slight and unfortunately, as we have already shown, this is the only plane of movement that is not unpleasant.

(2) The frontal plane—namely, a rolling of the ship from side to side. If the individual is standing facing the bow of the ship, the vertical canals are affected in the frontal plane. This is unpleasant. If, therefore, the individual lies down with his head or his feet toward the bow, the rolling movement then affects his horizontal canals, and the unpleasantness disappears.

(3) The sagittal plane—namely, a pitching of the ship fore and aft. If the individual is standing facing the bow of the ship, the vertical canals are affected in the sagittal plane. This is unpleasant. If, therefore, the person lies down with the line of the

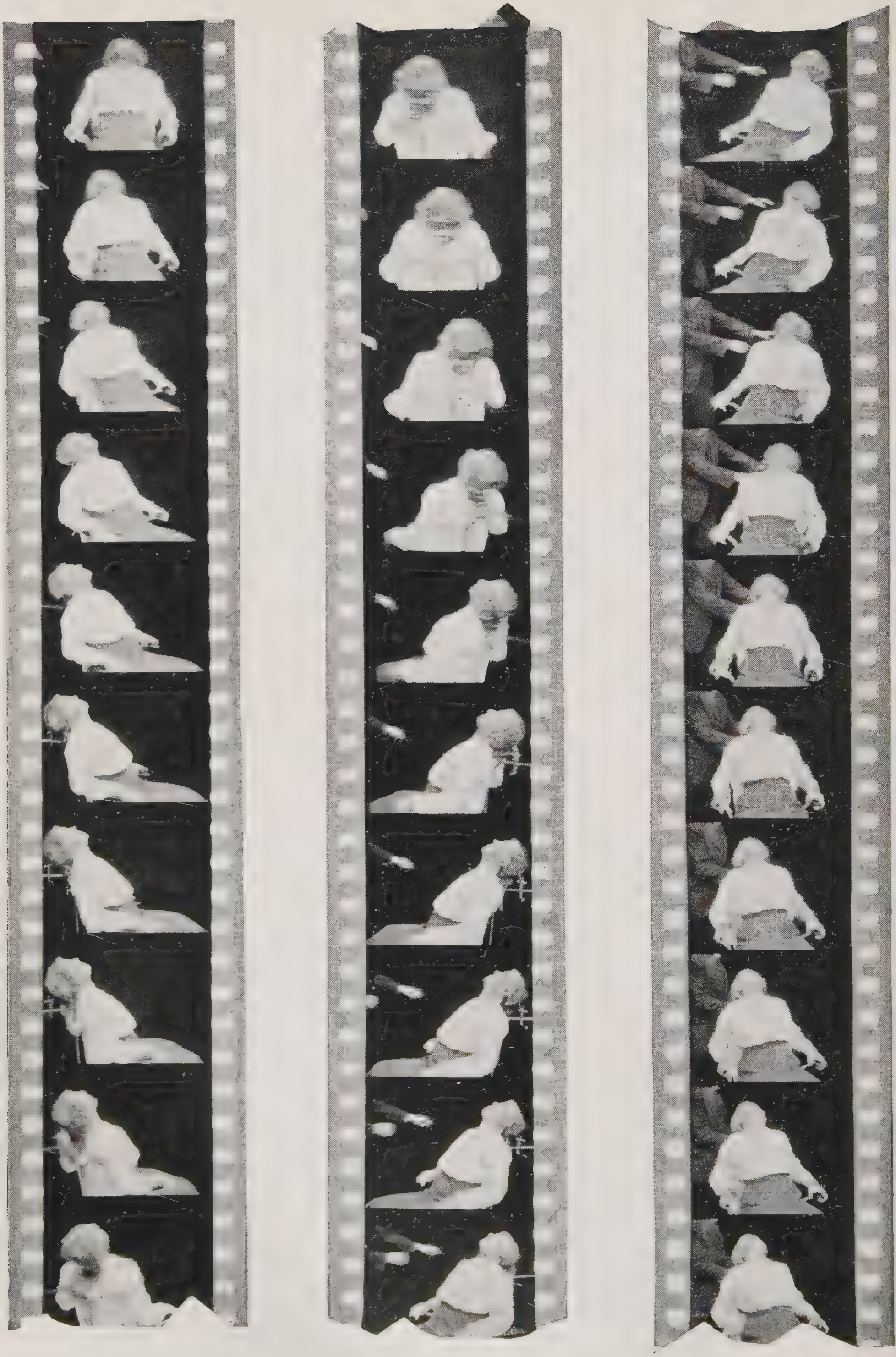


FIG. 50.—Turning to the left with the head 60° back.

body extending across the ship from starboard to port, the pitching movement then affects the horizontal canals, and the unpleasantness disappears.

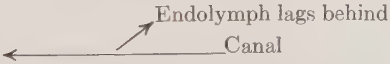
The up-and-down movement of the ship, rising and sinking, in a similar way affects the vertical canals when the individual is in the upright position. The unpleasantness caused by this movement is also relieved when the individual lies down, as then the up-and-down movement affects the horizontal canals, the stimulation of which is so much less unpleasant.

## The Artificial Production of Vestibular Vertigo

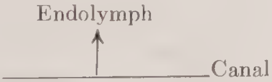
The subjective sensation of vertigo after ear stimulation is due to the movement in one direction or the other of the hair-cells of the end-organ in the labyrinth; such sensations can be produced either by turning an individual in a revolving chair or by douching the ears.

### Vertigo After Turning

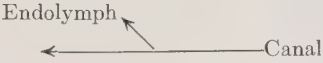
If a person is turned with the head upright toward the right, with eyes closed, his first sensation is of turning toward the right. This is due to the lagging behind of the endolymph in the horizontal semicircular canals. Diagramatically this can be represented as in preceding chapter, in explaining nystagmus:



Now as the turning is continued the endolymph catches up to the movement of the body and the individual no longer *feels* that he is turning, although actually he is turning.



On stopping the chair, the endolymph continues to move and the person has a sensation of turning in the opposite direction, namely to the left, although he is actually sitting absolutely quiet in the chair.



The essential feature of the subjective sensation of vertigo after turning is that after turning to the right the individual feels he is turning to the left, *regardless of the position of the head.* After turning to the right with the head forward, or after turning to the right with the head backward, although the movement of the endolymph is in a diametrically opposite direction, the result is the *same,* namely, a subjective sensation of turning to the left. It will be recalled that this is not true of nystagmus. Turning to the right with the head forward produces nystagmus to the *left;* turning to the right with the head backward produces nystagmus to the *right.* This is because nystagmus is a simple reflex depending directly on the direction of the endolymph movement. Vertigo, on the other hand, is a cerebral phenomenon; in determining the interpretation of the sensation of movement, the cerebrum *takes into consideration the position of the head* and in this way properly interprets the significance of the endolymph movement with the head in any given position.

The following is a table of the subjective sensations of vertigo produced by turning:

Turning to the right with the head upright produces a sensation of movement to the left in the horizontal plane.

Turning to the left with the head in the upright position produces a sensation of turning to the right in the horizontal plane.

Turning to the right with the head forward 120° produces: (*a*) With the head kept in the forward position, a sensation of turning to the left in the horizontal plane. (*b*) With the head brought upright, a sensation of falling to the left in the frontal plane.

Turning to the left with the head forward 120° produces: (*a*) With the head kept in the forward position, a sensation of turning to the right in the horizontal plane. (*b*) Bringing the head to the upright position, a sensation of falling to the right in the frontal plane.

Turning to the right with the head back 60° produces: (*a*) With the head kept in this backward position, a sensation of turning to the left in the horizontal plane. (*b*) With the head brought upright, a sensation of falling to the right in the frontal plane.

Turning to the left, head back 60°, produces: (*a*) With the head kept in this backward position, a sensation of turning to the right in the horizontal plane. (*b*) Head brought to upright position, a sensation of falling to the left in the frontal plane.

It is not necessary to memorize all the subjective sensations of turning. In the chapter on nystagmus it is noted that the eyes are always drawn in the direction of the endolymph movement. As regards the subjective sensation of systematized vertigo, the following rule should be remembered: *Vertigo is always in the direction opposite to the endolymph movement.*

Experience, through sight and muscle-sense, has taught the individual that when he turns to the right and the endolymph is lagging behind, that he is turning to the right. When he stops and the endolymph continues to go to the right, he therefore has a subjective sensation of turning to the left. The original basis for his mental interpretation of the endolymph is that when he turns to the right the endolymph moves relatively to the left. The mental interpretation, therefore, is always of a subjective sensation of movement in a direction opposite to the endolymph current.

## VERTIGO AFTER DOUCHING

On douching the ear with either cold or hot water, continued a sufficient time to permit a chilling or warming of the outer wall of the labyrinth, there occurs a systematized vestibular vertigo. The following is a table of the subjective sensations of vertigo produced by the caloric test:

Douching the right ear, head upright, water 68°, produces sensation of falling to the left in the frontal plane.

Douching the left ear with the head upright, water 68°, produces sensation of falling to the right in the frontal plane.

Douching the right ear with the head 120° forward, water 68°, produces sensation of falling to the right.

Douching the left ear with the head 120° forward, water 68°, produces sensation of falling to the left.

Douching the right ear, head back 60°, water 68°, produces sensation of falling to the left.

Douching the left ear, head back 60°, water 68°, produces sensation of falling to the right.

Douching the right ear, head upright, water 112°, produces sensation of falling to the right in the frontal plane.

Douching the left ear, head upright, water 112°, produces sensation of falling to the left in the frontal plane.

Douching the right ear, head 120° forward, water 112°, produces sensation of falling to the left.

Douching the left ear, head 120° forward, water 112°, produces sensation of falling to the right.

Douching the right ear, head back 60°, water 112°, produces sensation of falling to the right.

Douching the left ear, head back 60°, water 112°, produces sensation of falling to the left.

## CHAPTER XVII

## POINTING TESTS OF BARANY

In discussing the pointing tests it is first necessary to consider the functions of *orientation* and *equilibration*. While these two functions are co-related they are nevertheless not identical. By orientation is meant the determining of the relation of the body to space. By equilibration is meant the maintenance of position, whether walking, standing or sitting. "Pointing" is a cerebral act of orientation.

In 1909 Barany, while working on the physiology of the ear-tests, became interested in the pointing tests of von Graefe. These are tests for eye-muscle palsy. The patient is allowed to look at a fixed object with the suspected eye, keeping the normal eye closed. In other words, by means of the sight of the suspected eye, he fixes in his mind the supposed position of the external object. Then with both eyes shut, he attempts to point to the object with his finger. If he is able to touch it, it shows that there is no eye-muscle palsy. If he *past*-points, however, it shows that there is a palsy. This is because the eye, being out of the normal line, has given him false information as to the position of the external object. It then occurred to Barany that if the misplaced position of the eye-ball, due to palsy, would cause a past-pointing, the misplaced position of the eye during a *nystagmus* would also probably produce a past-pointing. This he found to be true. After the turning or douching, instead of being able to touch the external object, the individual pointed past the object, to one side or the other. On further examination, however, Barany found that with the *eyes closed* the past-pointing was present just the same as when the eyes were open; in other words, it was not an ocular phenomenon at all. Past-pointing after turning or douching is *not* due to the misplaced position of the *eyes* or to the disturbance of vision, but is due to the stimulation of the *ears*.

It is this contribution of Barany that was epoch-making in the study of the relation of the ears to the central nervous system. It is by means of these pointing-tests that we come to realize the intimate relation between the ears and every portion of the body-musculature through the nervous system.

Now as regards the pointing-tests themselves; the normal person is always aware of the location of his hand or finger in space. Furthermore, with his eyes closed he is aware of the exact position in space of objects previously located. Therefore, having first touched with his finger an object in space, he is able with the eyes closed to move his finger away from the object and then come back and find it again; and this, not as a mere approximation completed by groping for the object, but as a prompt, assured and exact return to the point previously touched.

## Spontaneous Pointing Tests

The spontaneous tests consist of determining the patient's ability, with the eyes shut, to find the finger of the examiner which he has previously felt. All normal individuals are always able to find the finger almost or quite as readily as if the eyes were open.

Shoulder from above (Fig. 51) shows the upper extremity of the patient extended straight in front of him. His fist is closed with the exception of the forefinger, which is extended straight in front of him. He is blindfolded; this is not necessary, provided we are sure that the patient can be trusted to keep his eyes closed. The examiner places the finger of the patient directly above his own forefinger. The patient is told to fix in his mind the exact position of the examiner's finger. The examiner then says "Up." The patient raises his arm to the vertical position and then immediately brings it back again to find the examiner's finger. Fig. 52 shows the same test, but the moving pictures were taken from above and behind the patient, in order to show more clearly that there is no lateral deviation of his finger.

Shoulder from below (Fig. 53). The patient's forefinger is placed immediately underneath the forefinger of the examiner, and

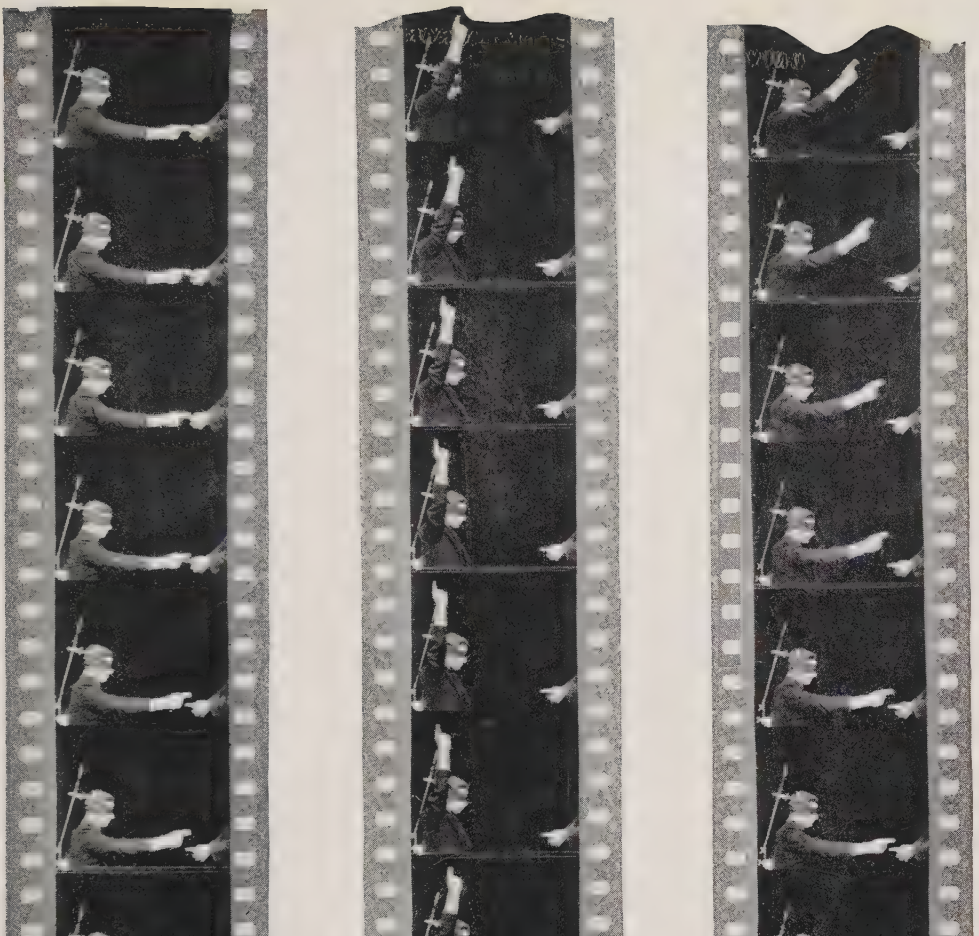
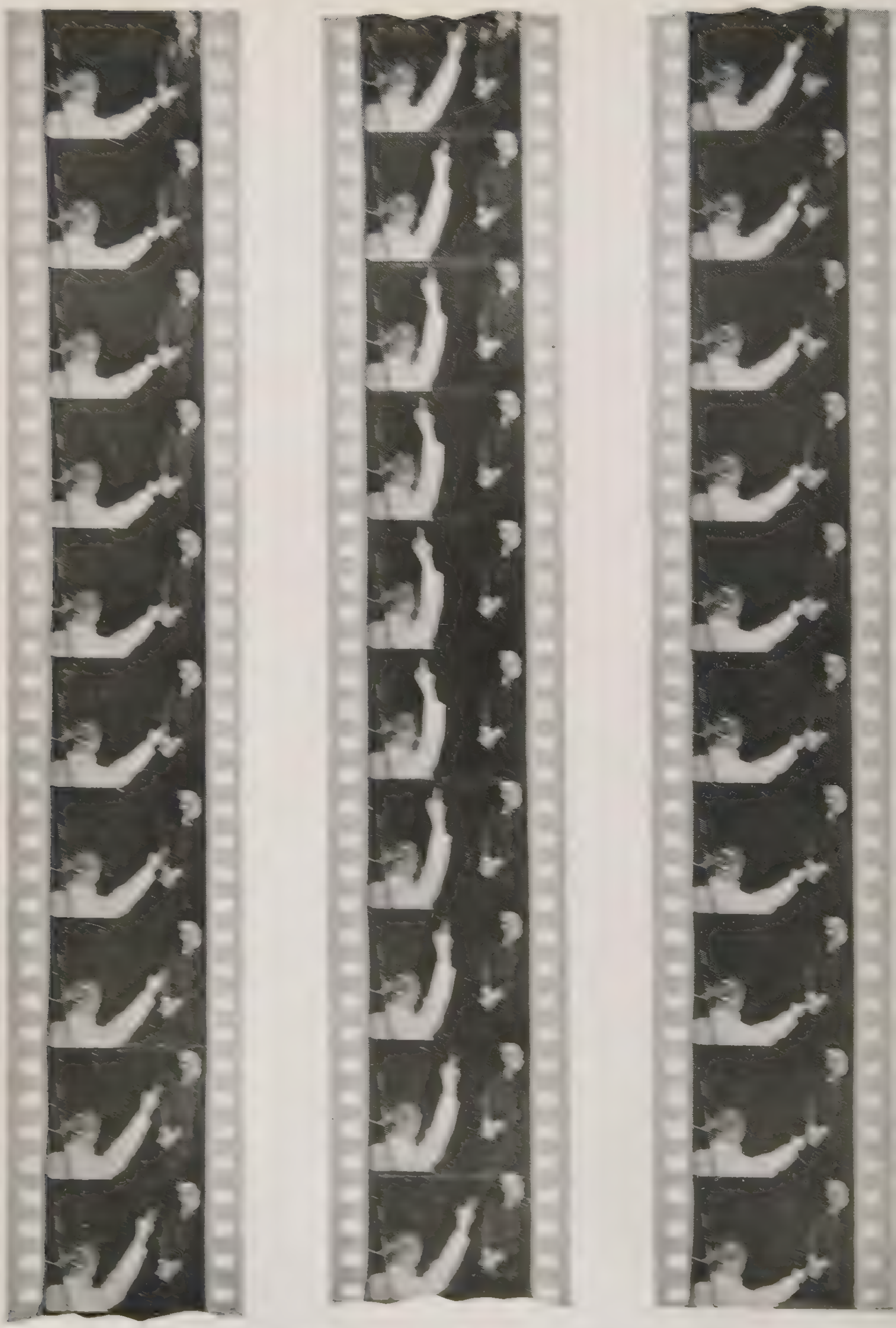


FIG. 51.—Shoulder from above.



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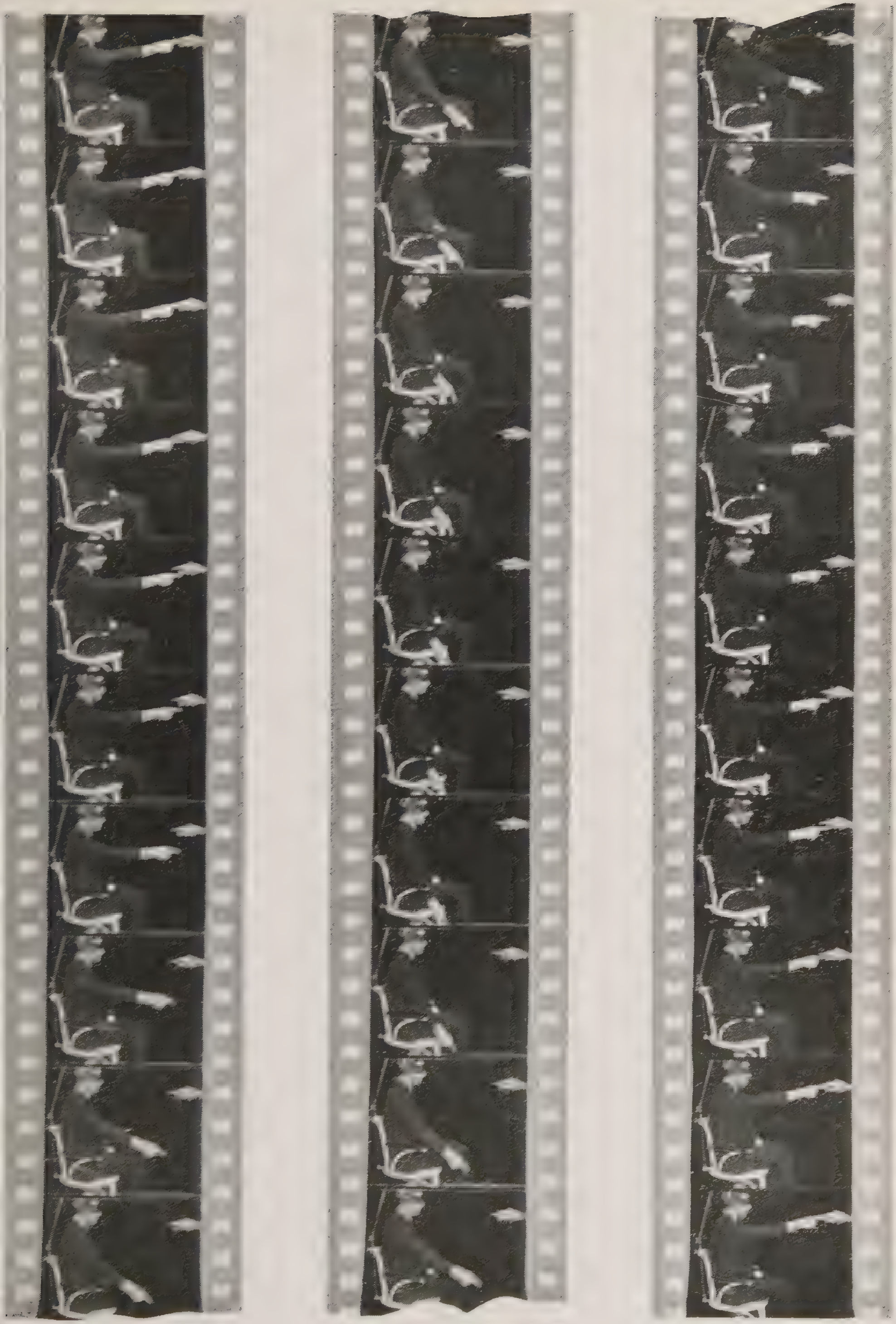


[illegible]

the examiner says "Down." The patient slowly lowers his arm and immediately brings it back again to touch the finger of the examiner.

Shoulder from the side (Fig. 54). The patient's finger is placed alongside of the finger of the examiner. The examiner says "Out," the patient brings his arm out sideways and immediately brings it back to find the finger of the examiner.

Shoulder to the side (Fig. 55). The patient touches the examiner's finger in the side position, brings his arm horizontally forward to the middle line and immediately brings it back and finds the finger of the examiner.

Elbow from below (Fig. 56). Only the forearm must be used in this movement. The arm is therefore held firmly by an assistant, as shown in the moving pictures. The examiner places his own right forefinger at a point near the patient's head, and as this position is somewhat awkward for the examiner it is best for him to support his right hand with his left hand, as shown in the pictures. The patient's finger is placed just in front of the examiner's finger and the examiner says "Down." The patient lowers the forearm to the horizontal position and then brings it back and finds the finger of the examiner.

Elbow from above (Fig. 57). The patient's arm is held by an assistant; the examiner places his forefinger beneath the forefinger of the patient and says "Up." The patient raises his forearm as high as possible and immediately brings it back again to find the finger of the examiner.

Wrist from below (Fig. 58). As the hand alone should move in this test, an assistant holds the forearm firmly. The examiner places his forefinger in approximately the vertical position, and places the patient's finger in front of it. The patient lowers his finger as far as possible and immediately brings it back to find the finger of the examiner.

Hip from below (Fig. 59). The patient elevates the lower extremity in a straight line without bending the knee. The examiner places his forefinger on the big toe or on a certain spot of the shoe. The examiner says "Down." The patient lowers the foot, with-

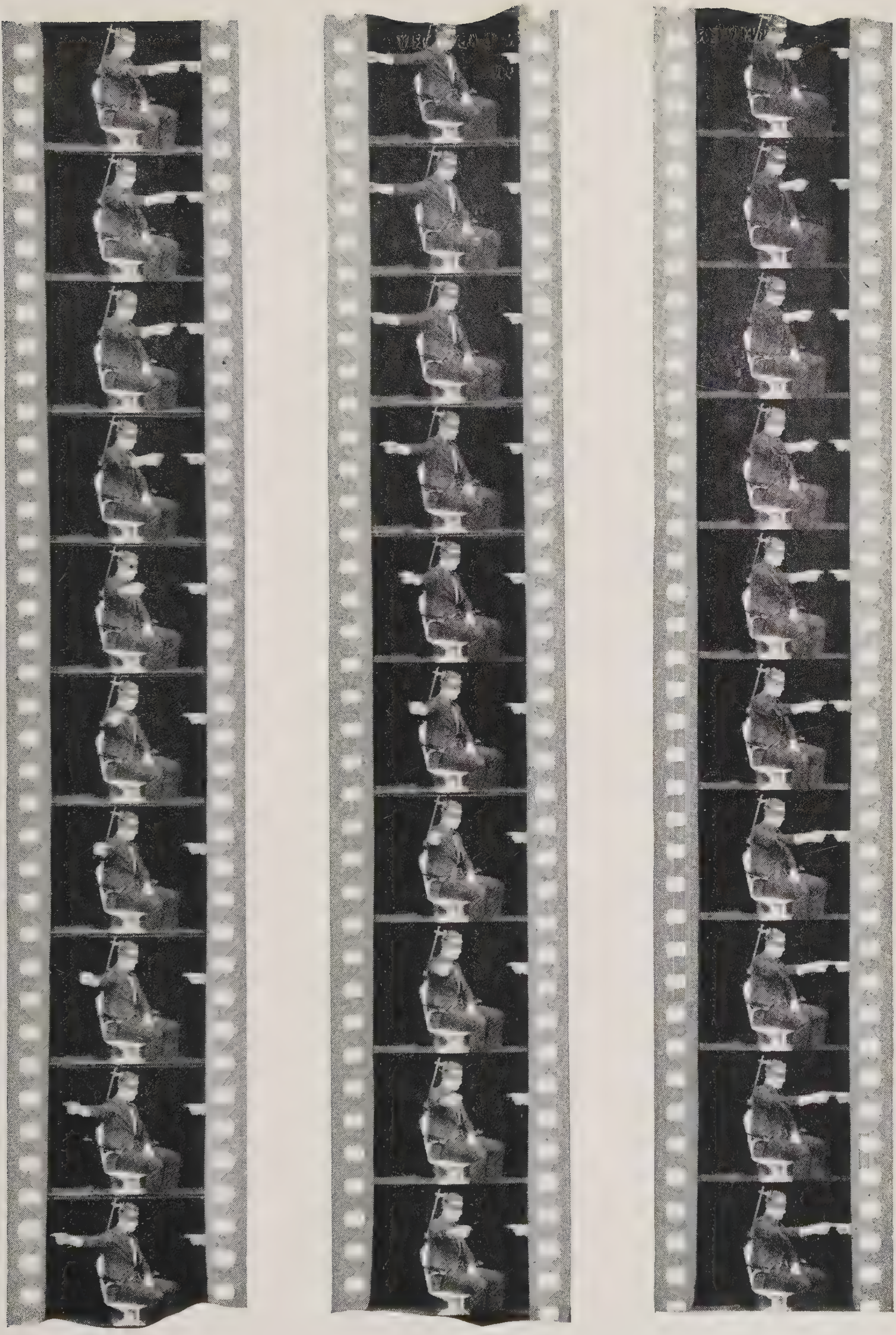


FIG. 54.—Shoulder from the side.

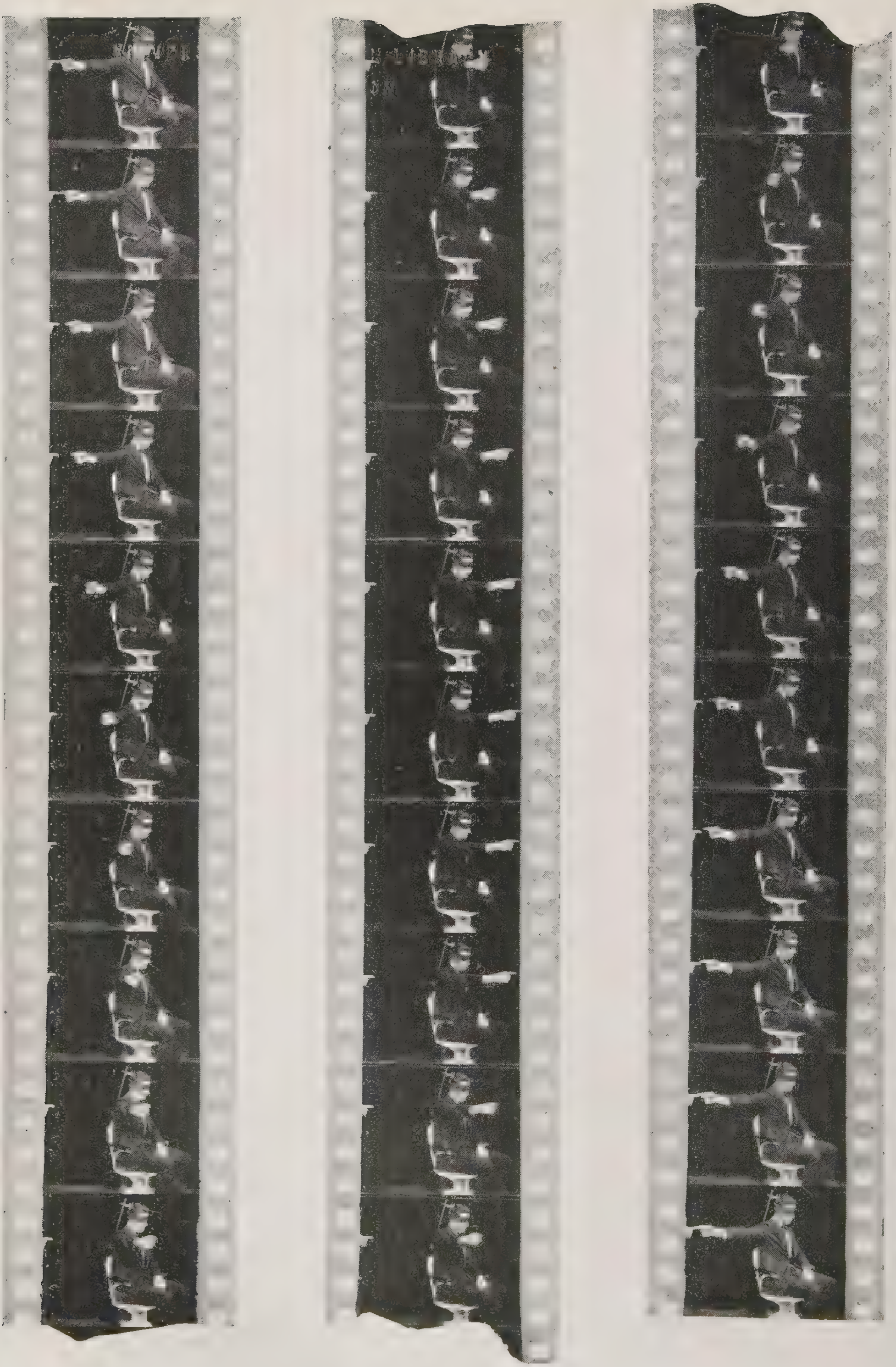


Fig. 55.—Shoulder to the side.

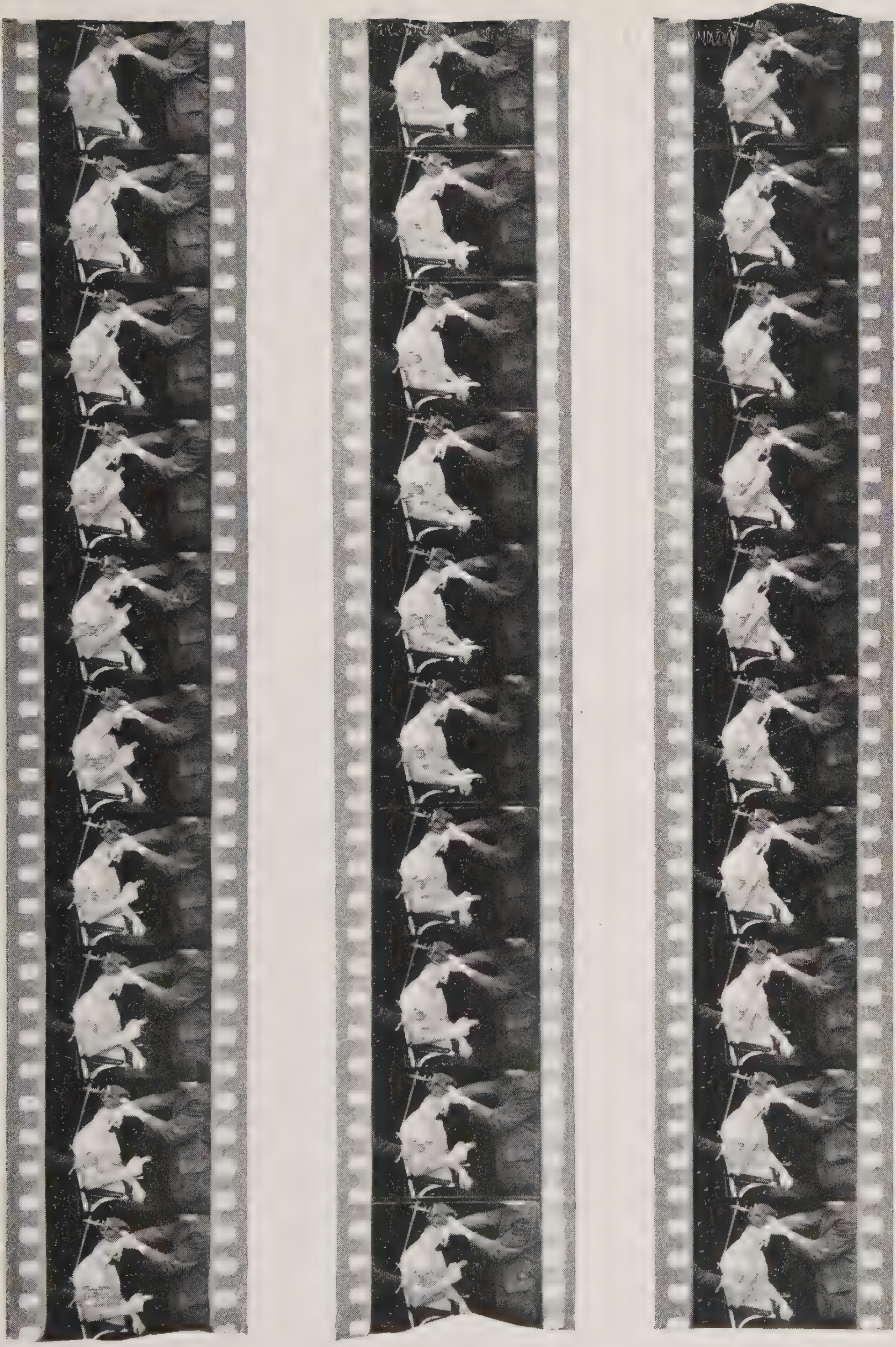


Fig. 56.—Elbow from below.

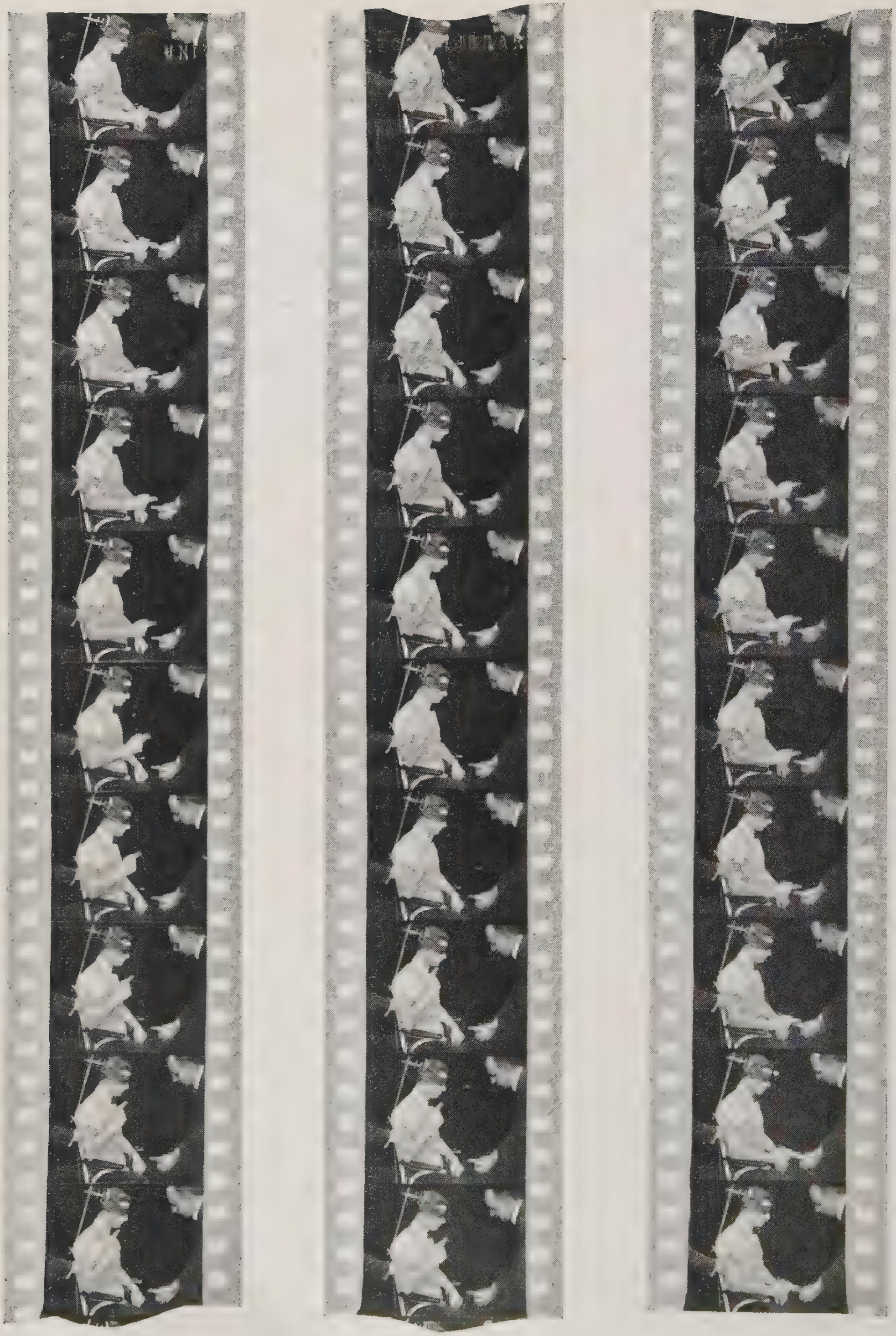


FIG. 57.—Elbow from above.

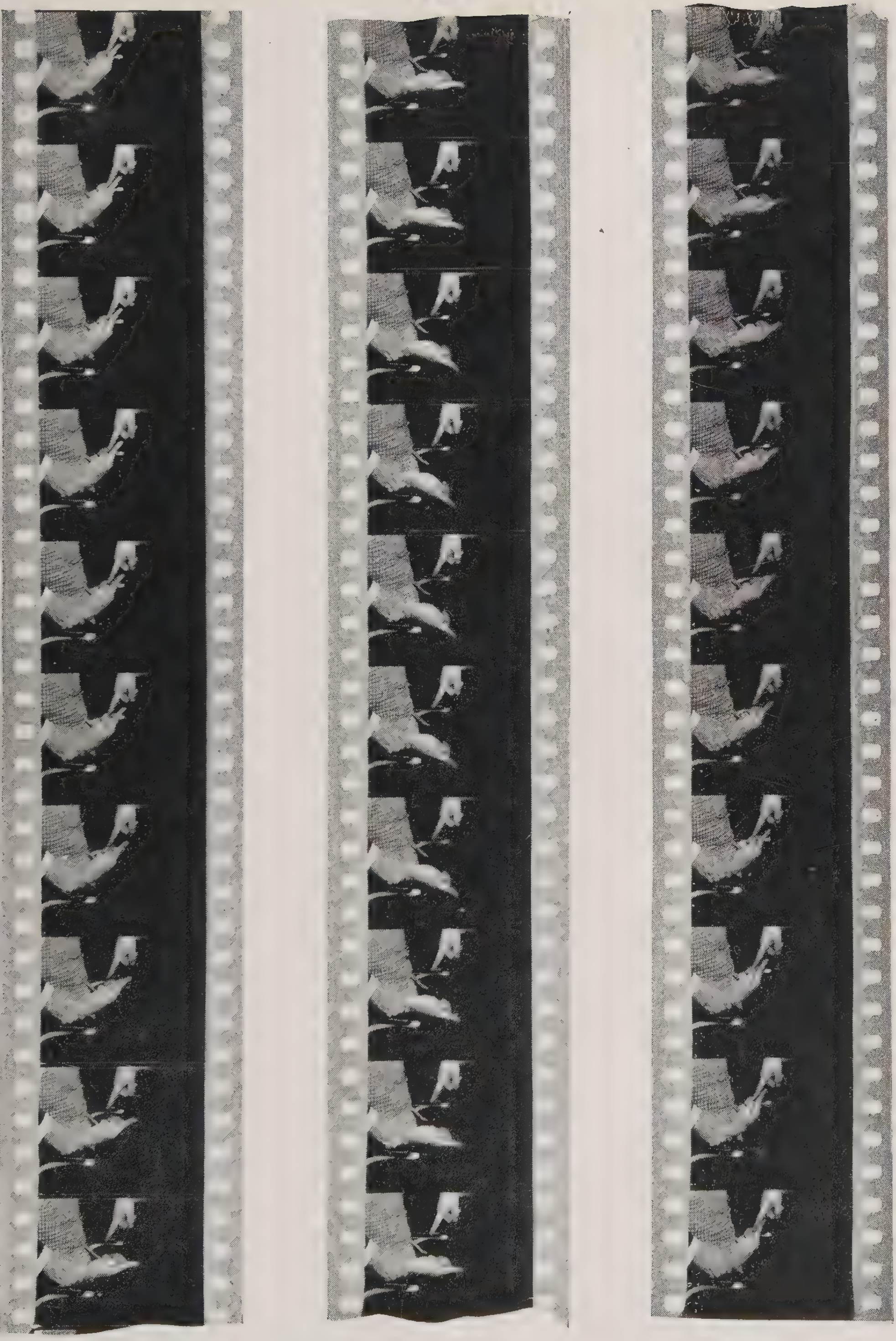


Fig. 58.—Wrist from below.

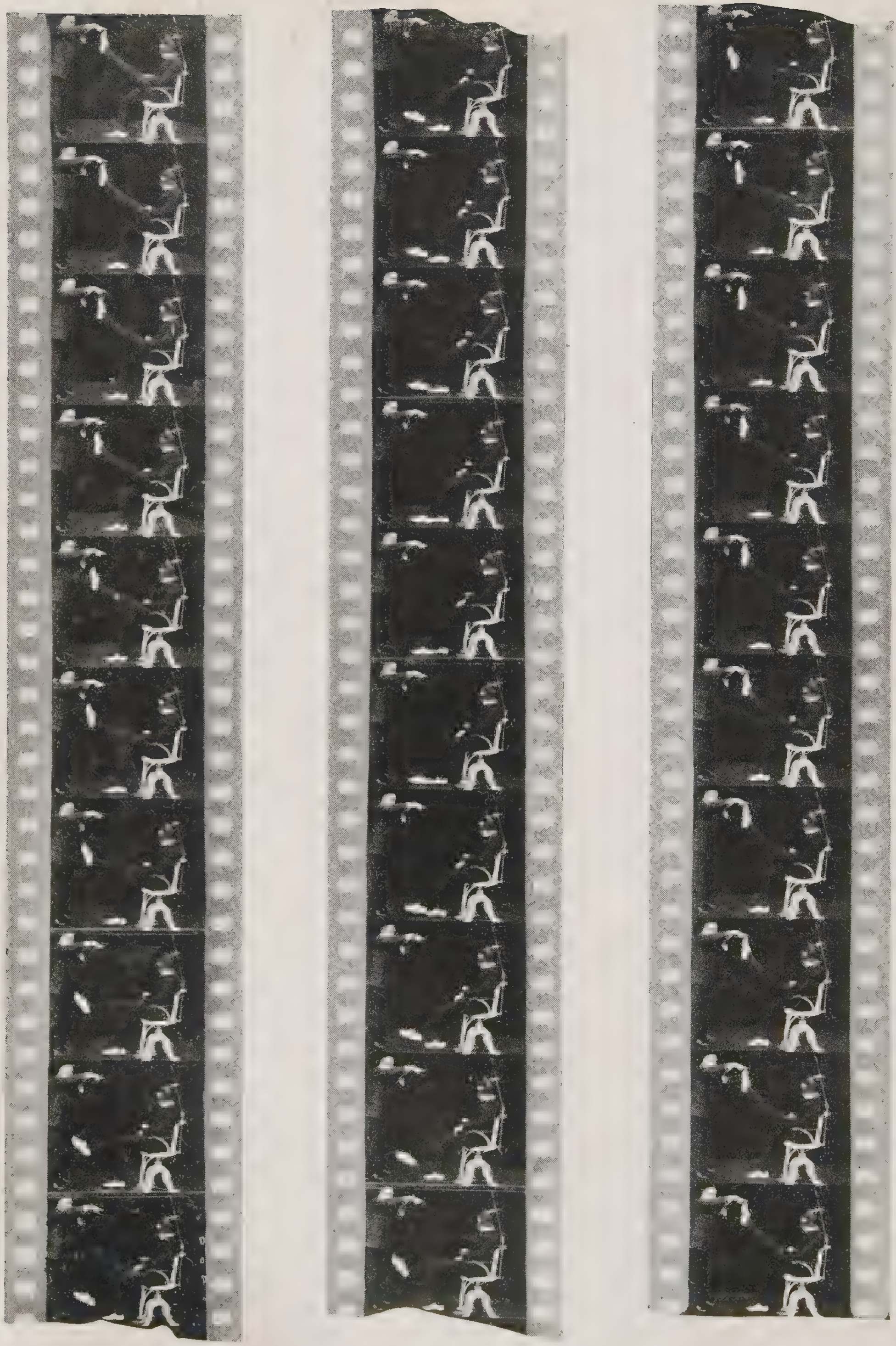


FIG. 59.—Hip from below.

out bending the knee, and brings it back again to find the finger of the examiner.

Neck from below (Fig. 60). In order to make an accurate test of any lateral movement of the head, a pointing instrument is used; a lead-pencil attached to an ordinary head-band is satisfactory. The examiner places his forefinger directly back of the lead-pencil, and says "Down." The patient bends the head forward and attempts to bring it back directly in a straight line. The lead-pencil will then touch again the finger of the examiner. In testing the neck it is essential that the body shall not move. Therefore, as shown in the pictures, the shoulders of the patient are held firmly by an assistant.

Trunk from below (Fig. 61). The examiner places his finger directly back of the pointing instrument, and says "Down." The patient bends his entire body forward, and attempts to bring it back again in a straight line. The pointing instrument is brought back to touch the finger of the examiner.

In all of these spontaneous pointing tests, the normal individual is invariably able to find the finger of the examiner. If the doctor does not speak the language of the patient, he can explain the tests in pantomime, with the patient's eyes open, and then conduct the tests with the eyes closed.

## Pointing Tests After Ear-stimulation

Now if the ear is stimulated either by turning the patient or douching his ear, and the above tests are again repeated, the patient, because of the systematized vertigo produced is no longer able to find the finger but "past-points." If he feels he is turning to the left he will past-point to the right; if he feels he is turning to the right he will past-point to the left.

## Past-pointing After Turning

The pictures (Fig. 62) were taken after turning the patient to the right with the head upright. The pictures were taken from above and behind the patient. The first picture shows the exam-

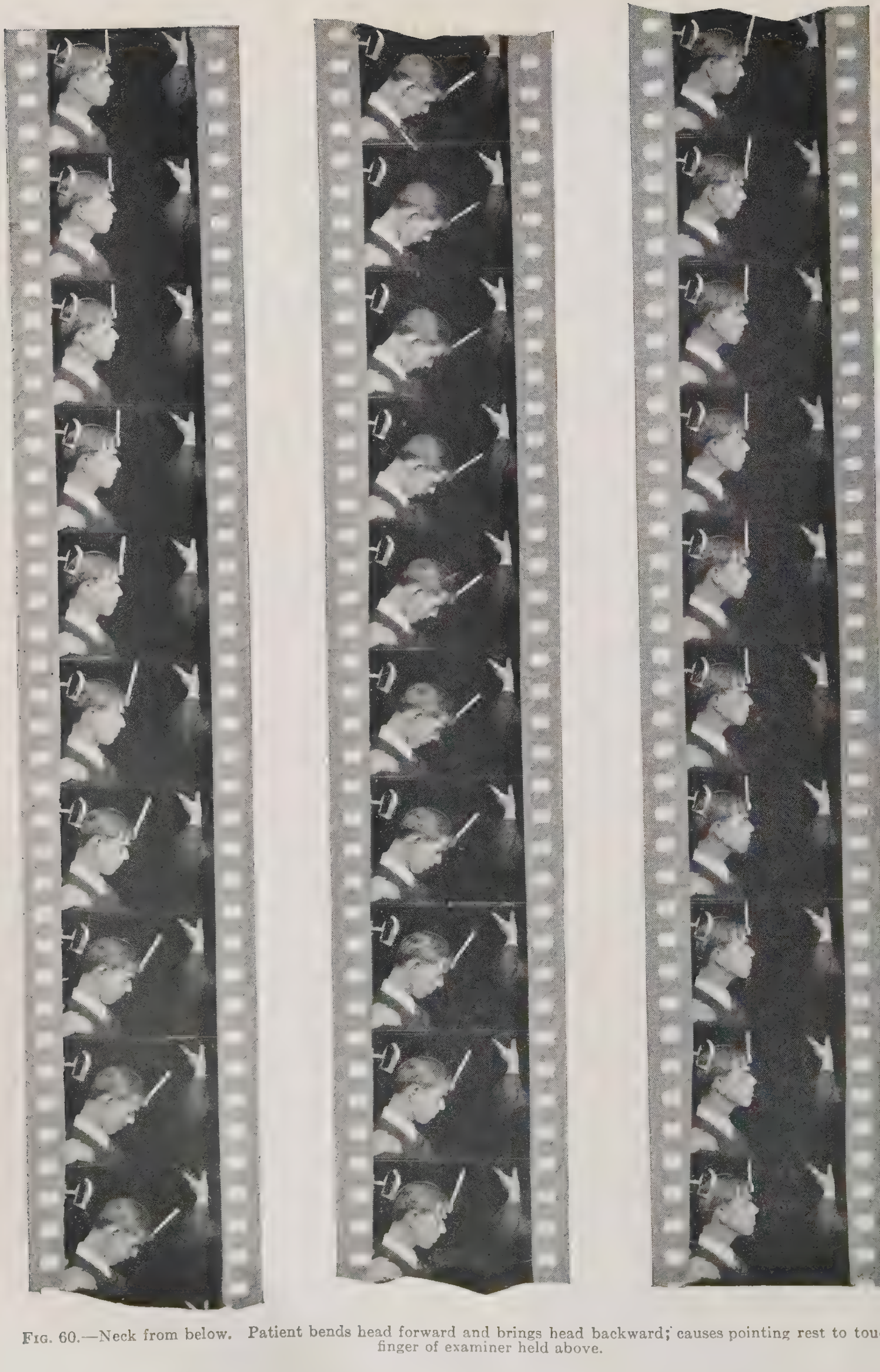


Fig. 60.—Neck from below. Patient bends head forward and brings head backward; causes pointing rest to tou finger of examiner held above.

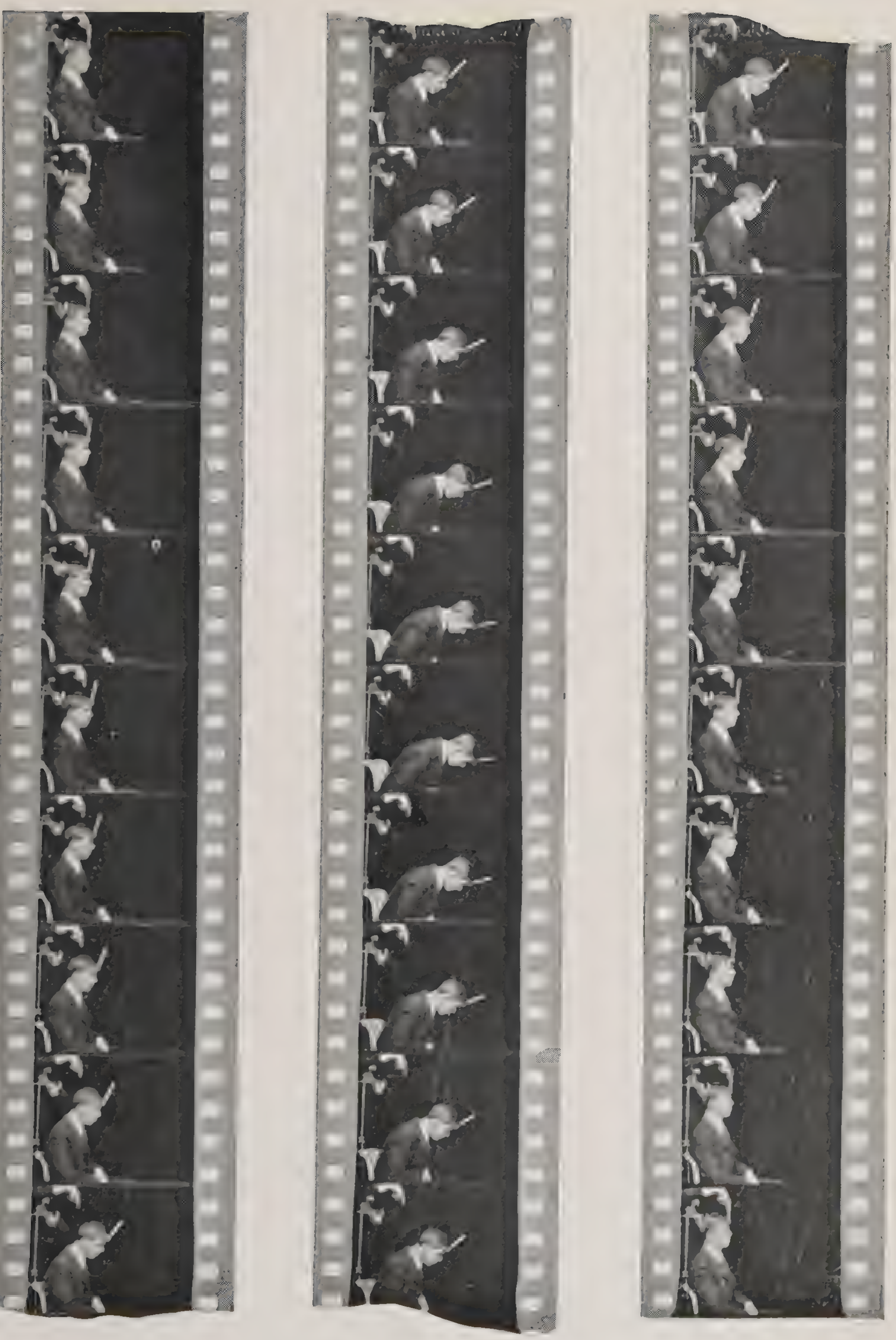


FIG. 61.—Trunk from below.

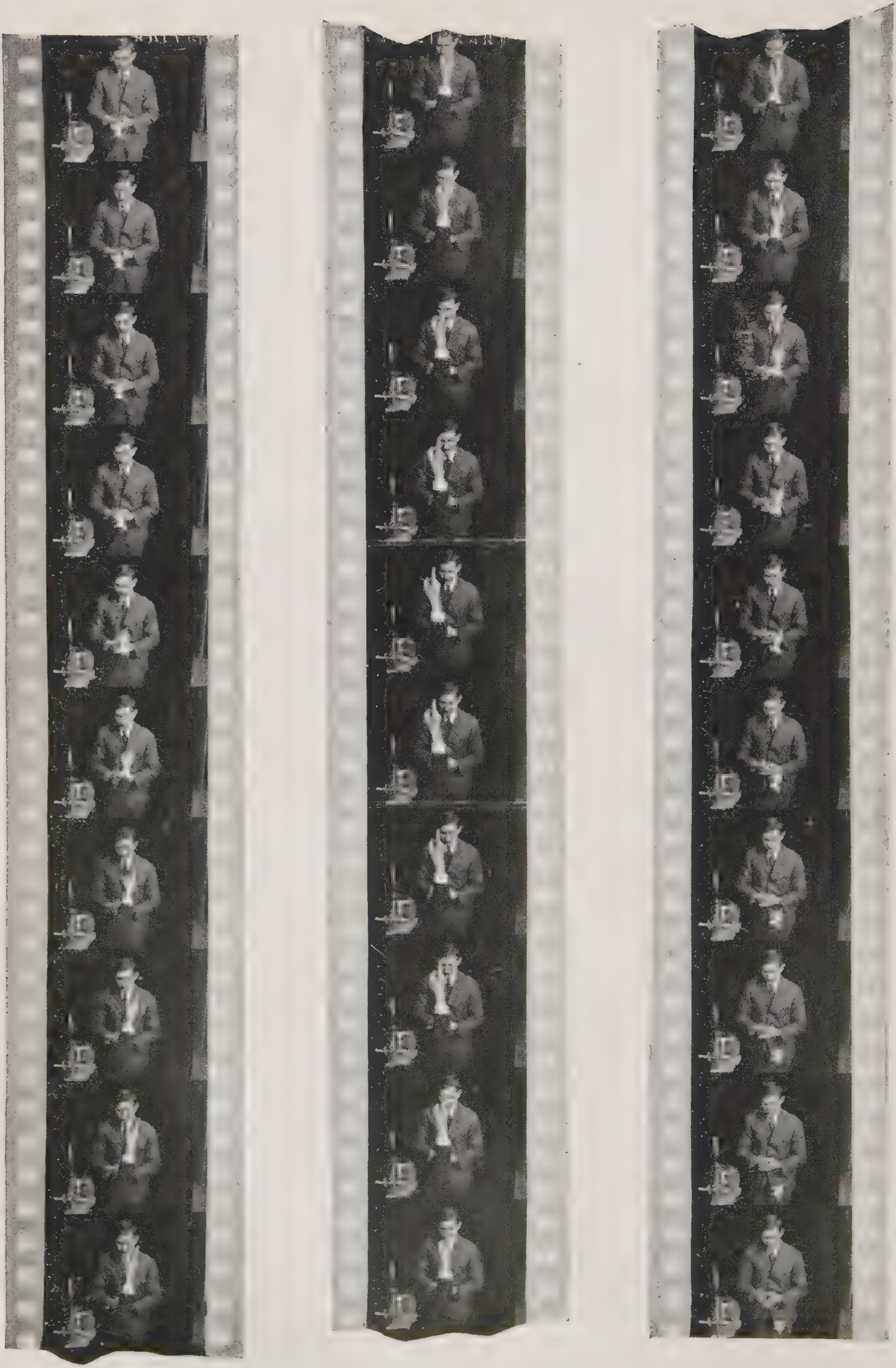


FIG. 62.—After turning to right, head 30° forward, endolymph movement in horizontal canal is to the right; therefore patient past-points to right.

iner touching the patient's finger immediately after the chair has been stopped. The patient gradually raises the arm and on bringing it back again, he is unable to find the doctor's finger but past-points to the right.

The pictures (Fig. 63) were taken after turning the patient to the left with the head upright. The first picture shows the arm being elevated; the patient continues to raise his arm to the vertical position and on coming back is unable to find the doctor's finger but past-points to the left.

## Past-pointing After Caloric Test

The pictures (Fig. 64) were taken after douching the right ear with cold water with the head upright. After the douching, the examiner has seized the patient's hand; the first picture shows the arm in the upright position. On coming back to find the finger, the patient is unable to do so but past-points to the right.

The pictures (Fig. 65) were taken after douching the patient's right ear with the head 60° backward. The patient feels the doctor's finger, raises the arm to the vertical position and on returning is unable to find the doctor's finger, but past-points to the right.

The pictures (Fig. 66) were taken after douching the left ear with cold water. The patient touches the doctor's finger, elevates the hand to the vertical position and on returning is unable to find the examiner's finger but past-points to the left.

The pictures (Fig. 67) were taken after douching the left ear with cold water with the head back 60°. The patient touches the doctor's finger, raises the hand to the vertical position and on returning is unable to find the finger but past-points to the left.

## Explanation of Past-pointing

Past-pointing is not an ear-*reaction.* Ear stimulation produces two reactions and two only. They are (1) nystagmus, and (2) vertigo.

Ear stimulation causes a pulling of the eyes; this is easily under-

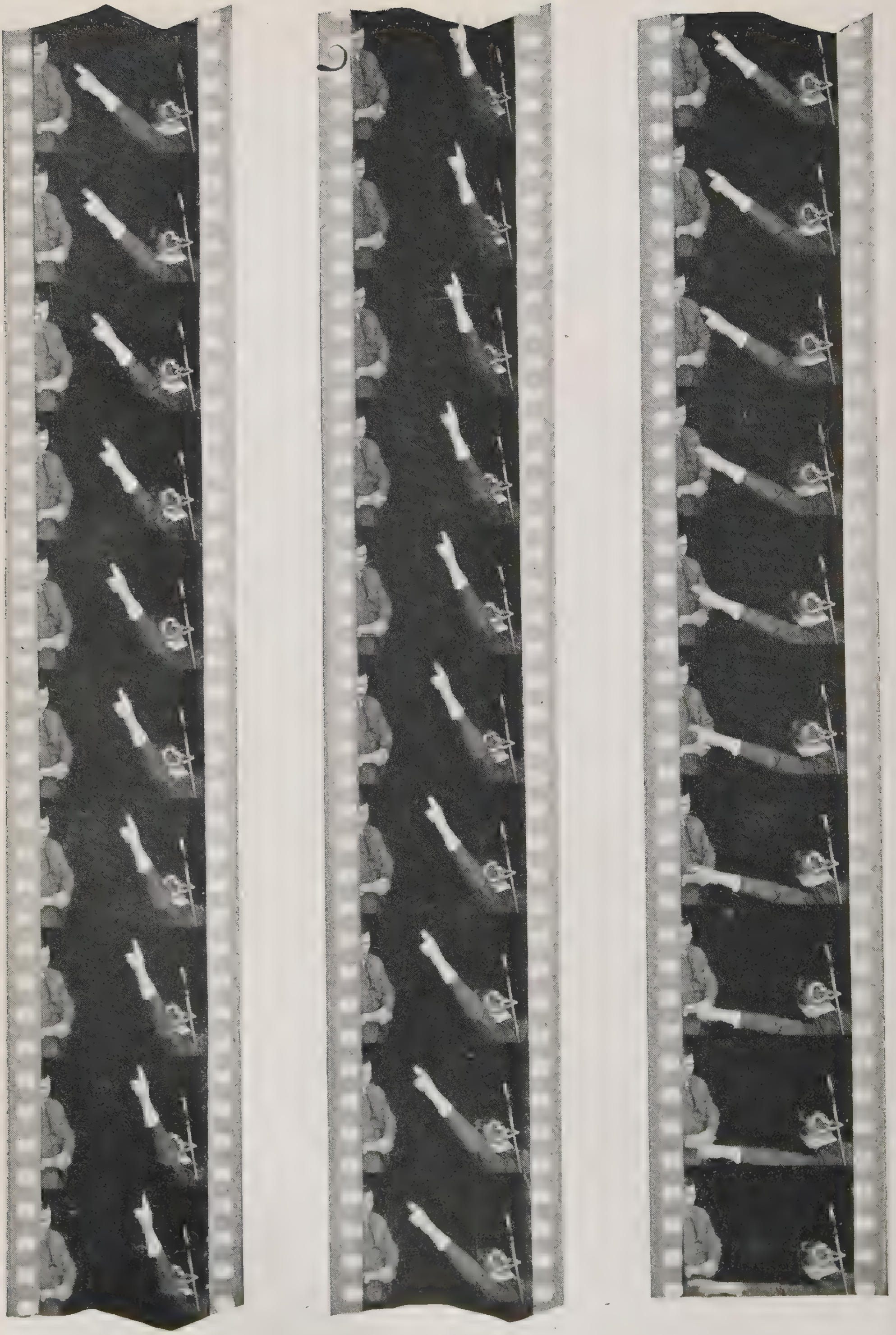
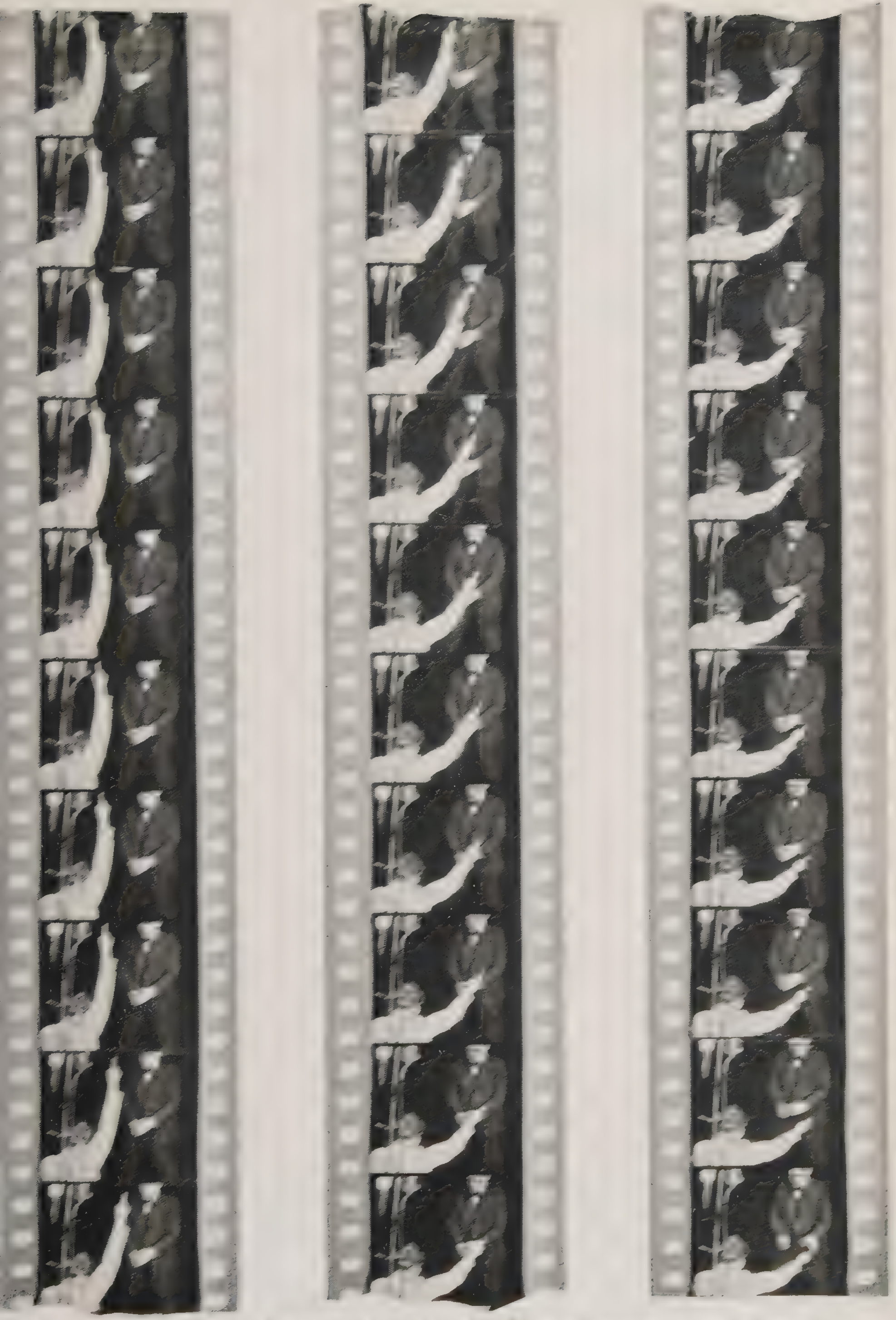


FIG. 63.—After turning to left, patient past-points to left.



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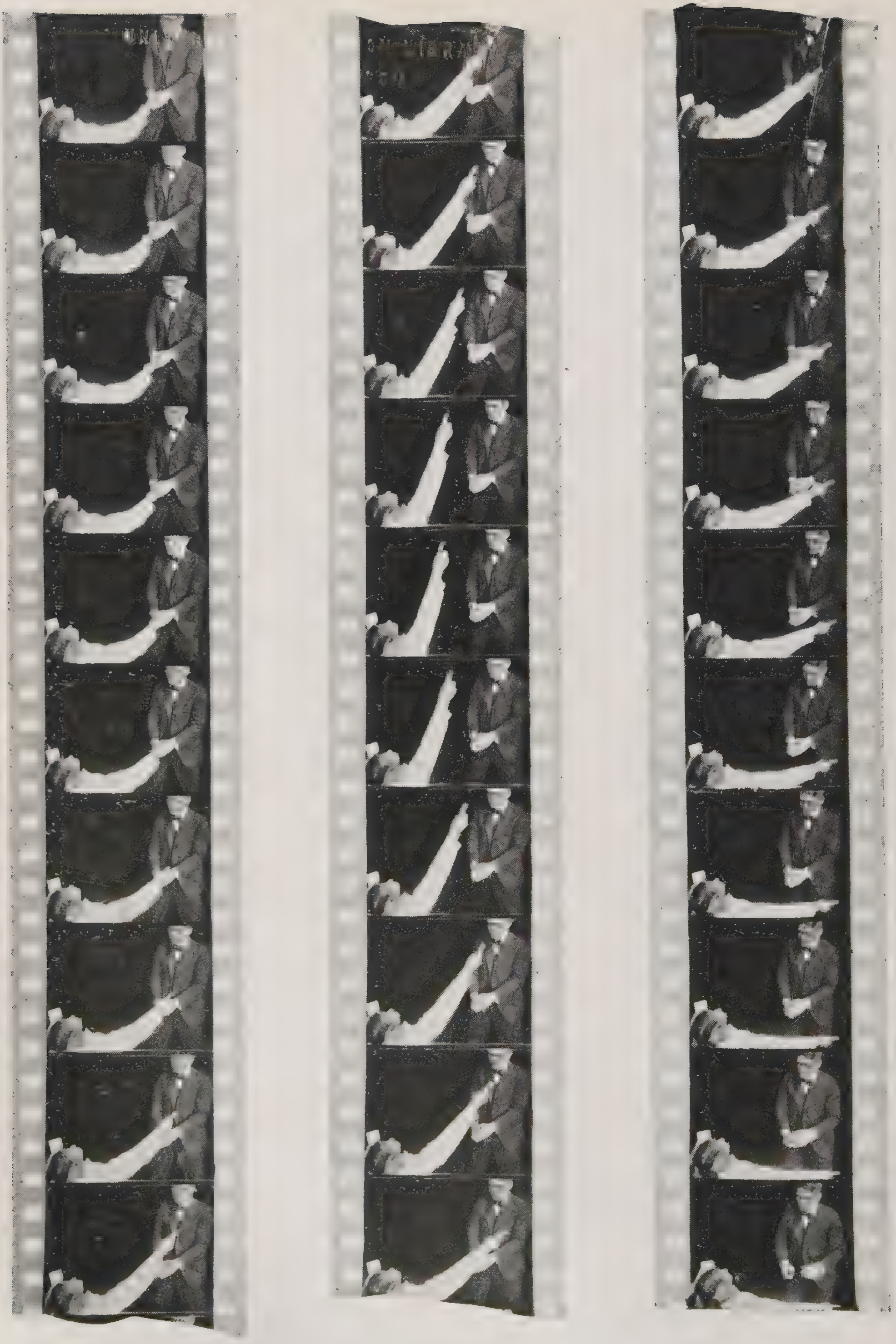

Fig. 65.—After douching right ear, water 68°, head 60° back, endolymph movement in horizontal canal is down to the right; therefore patient past-points to right.

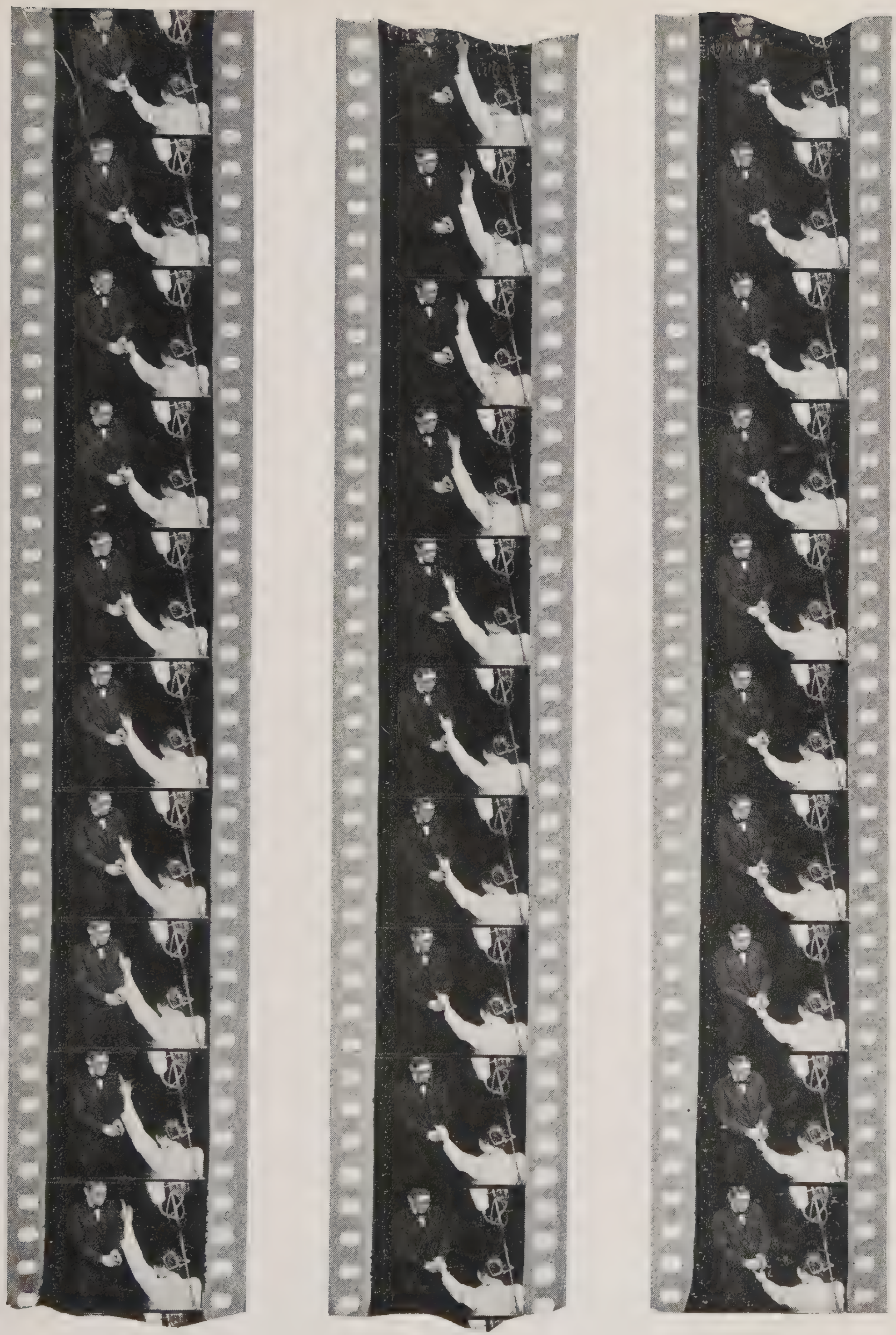

FIG. 66.—After douching left ear, water 68°, the endolymph movement is down to the left; therefore patient past-points to the left.

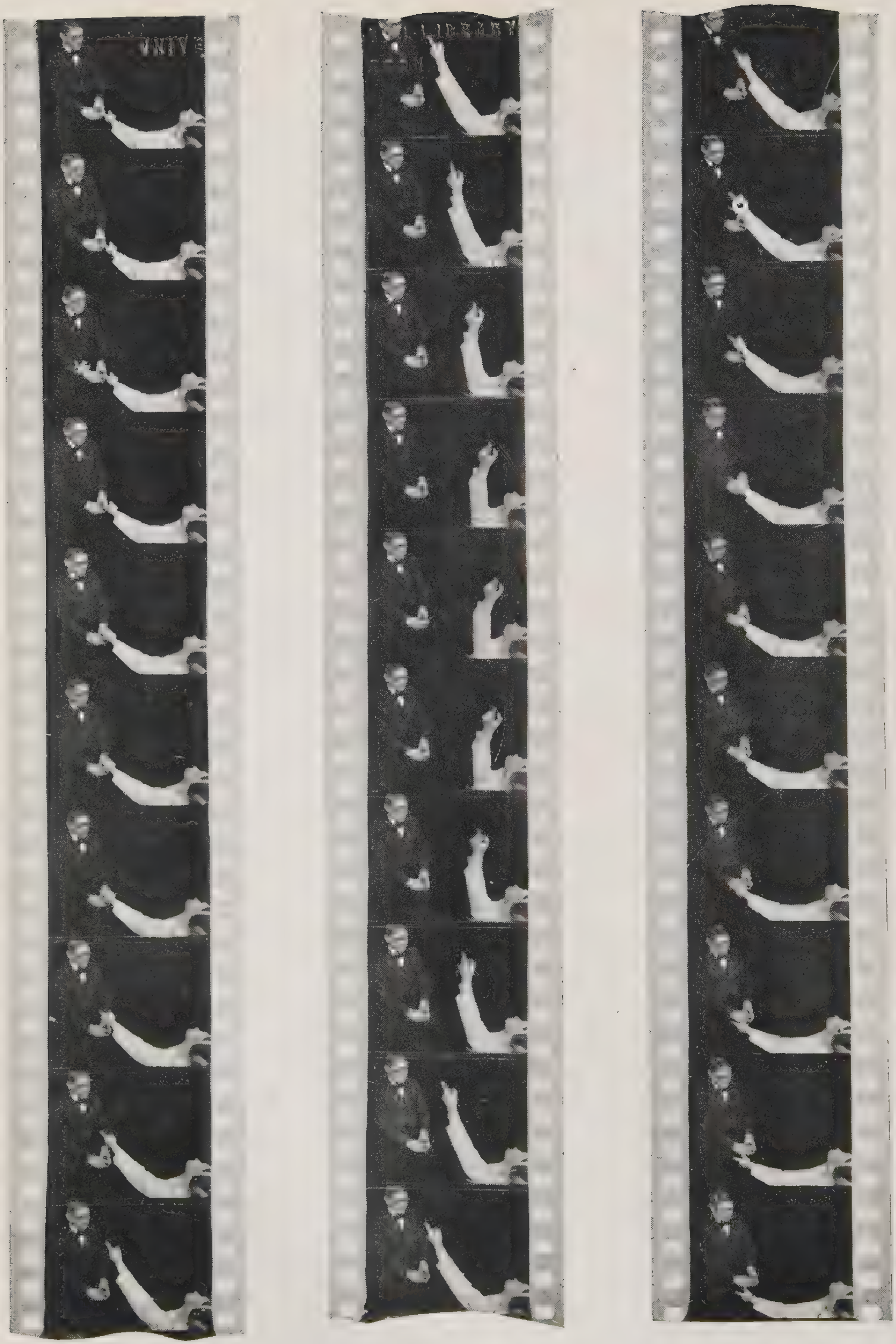

FIG. 67.—After douching left ear, head 60° back, endolymph movement in left horizontal canal is to left; therefore patient past-points to left.

stood as a simple *reflex*. The afferent sensory impulse is from the semicircular canals to the eye-muscle nuclei in the pons; the efferent motor impulse is from the eye-muscle nuclei to the eye muscles themselves. This reflex exists by itself and has no part in the causation of past-pointing. The other reaction to ear stimulation is vertigo. It is not a reflex; it is a subjective disturbance of the cerebral cortex due to sensory impulses received directly from the ear.

Pointing, on the other hand, is a cerebral motor act. Neither turning a person in a chair nor douching his ears causes him to past-point. *He is asked* to raise his arm and then bring it back to find the finger. Before ear stimulation he is able to find the finger; after ear stimulation he is unable to find the finger *because of the vertigo.* It may be regarded as a law of the ear-tests that where *there is no vertigo there is no past-pointing.* Vertigo is the primary reaction; past-pointing is a secondary manifestation. This is contrary to the general conception that past-pointing is due to a cerebellar *pull* of the arm to one side, in the same way that the eyes are drawn to one side by the stimulus from the labyrinth. Past-pointing is not a cerebellar function; it is cerebral. Spontaneously, the cerebral mandate is to find the finger at the exact point where it is; after ear-stimulation the cerebral mandate is to find the finger at a point where the cerebrum now *conceives* it to be. In either case the cerebellum through its synergic function, carries out the cerebral command with *accuracy;* the cerebellum inhibits wayward movement and by so doing makes possible the accurate co-ordinate act.

That past-pointing is not due to a cerebellar pull can be proven by the following five simple experiments:

(1) Anyone wishing to understand this phenomenon need merely have himself turned in the chair. After he is turned to the right he has, on stopping, a subjective sensation of turning to the left. He feels that he is leaving the examiner behind and *for this reason* he consciously and deliberately points to the right, where he conceives the finger now to be. If after the usual past-pointing, the examiner's finger is moved out to meet that of the

examined, and if the pointing is then repeated, he past-points still further to the right. He would continue perhaps to the completion of an entire circle in his chase for the seemingly moving finger, but for the limitations enforced by his seating. After experiencing this test, anyone is convinced that he has past-pointed because of his vertigo.

(2) An intelligent subject who knows that he would naturally past-point after stimulation, can voluntarily by a careful mental calculation, correct the past-pointing and deliberately point in the other direction. If he knows that he would naturally point twelve inches toward the right, he can deliberately point twelve inches toward the left and in this way may be able to touch the finger. This proves conclusively the mental control over the direction of the pointing.

(3) If the duration of vertigo, after turning, is timed with a stop-watch, and then a second test is immediately repeated, and followed by past-pointing, the duration of the past-pointing also being recorded by the stop-watch, it will be found that the duration of the vertigo and the duration of the past-pointing are the same. The average length of time in which the vertigo continues is 26 seconds and the average length of time in which the past-pointing continues is also 26 seconds.

(4) S. A. Brumm has demonstrated that after turning, if the individual is asked to move his finger up and down in a vertical plane, he is able to do so and does not deviate to the side. He merely moves his finger up and down in front of him in the vertical plane which he conceives to be revolving with him. If, however, he touches some *external object*, and then tries to find it again, he then feels that he is leaving that object behind him and past-points in that direction.

(5) Turning to the right with the head back 60° produces past-pointing to the right; turning to the right with the head forward 120° also produces past-pointing to the right. If past-pointing were due to a cerebellar pull this result would obviously be impossible; turning with the head backward and turning with the head forward produce diametrically opposite stimuli. If past-

pointing were a cerebellar reflex, the individual would in one instance point to the right and in the other instance point to the left. The past-pointing which occurs to the right in both instances, is because the cerebral centres receive the *same* impression of *vertigo*—that the body is turning to the left—no matter in what position the head may be placed.

The pictures (Figs. 68, 69) show the patient being turned to the right with the head back 60°. The examiner then takes the patient's hand and has him touch the finger. The patient raises the arm to the vertical position and on coming back is unable to find the finger but past-points to the *right*.

The pictures (Figs. 70, 71) show the patient being turned to the left with the head 60° back. On stopping the chair, the examiner seizes the patient's finger. The patient raises the arm to the vertical position and on bringing it back is unable to find the finger but past-points to the *left*. An individual will always past-point to the right when he is turned to the right, no matter whether the head is forward, upright or back. He will past-point to the left when he is turned to the left, no matter whether the head is forward, upright or back. It is evident therefore, that past-pointing is not a reflex but is a deliberate cerebral act.

Summarizing therefore, the cerebellum plays only a partial rôle in the large mechanism of the pointing. The kinetic-static sense, the arthrodial sense, tactile, auditory and visual impressions and memory all combine to inform the individual of the position of an external object. The motor areas of the cerebral cortex then send impulses to the arms; the function of the cerebellum is merely in controlling the execution of the cerebral mandate. If this be erroneous, because of the vertigo, past-pointing results.

## TRACTS FOR POINTING

The paths for the pointing and the paths for the past-pointing are one and the same. The mechanism of the pointing is the same whether the individual attempts to find the finger where it really is, or when he points in a false direction because of his vertigo.

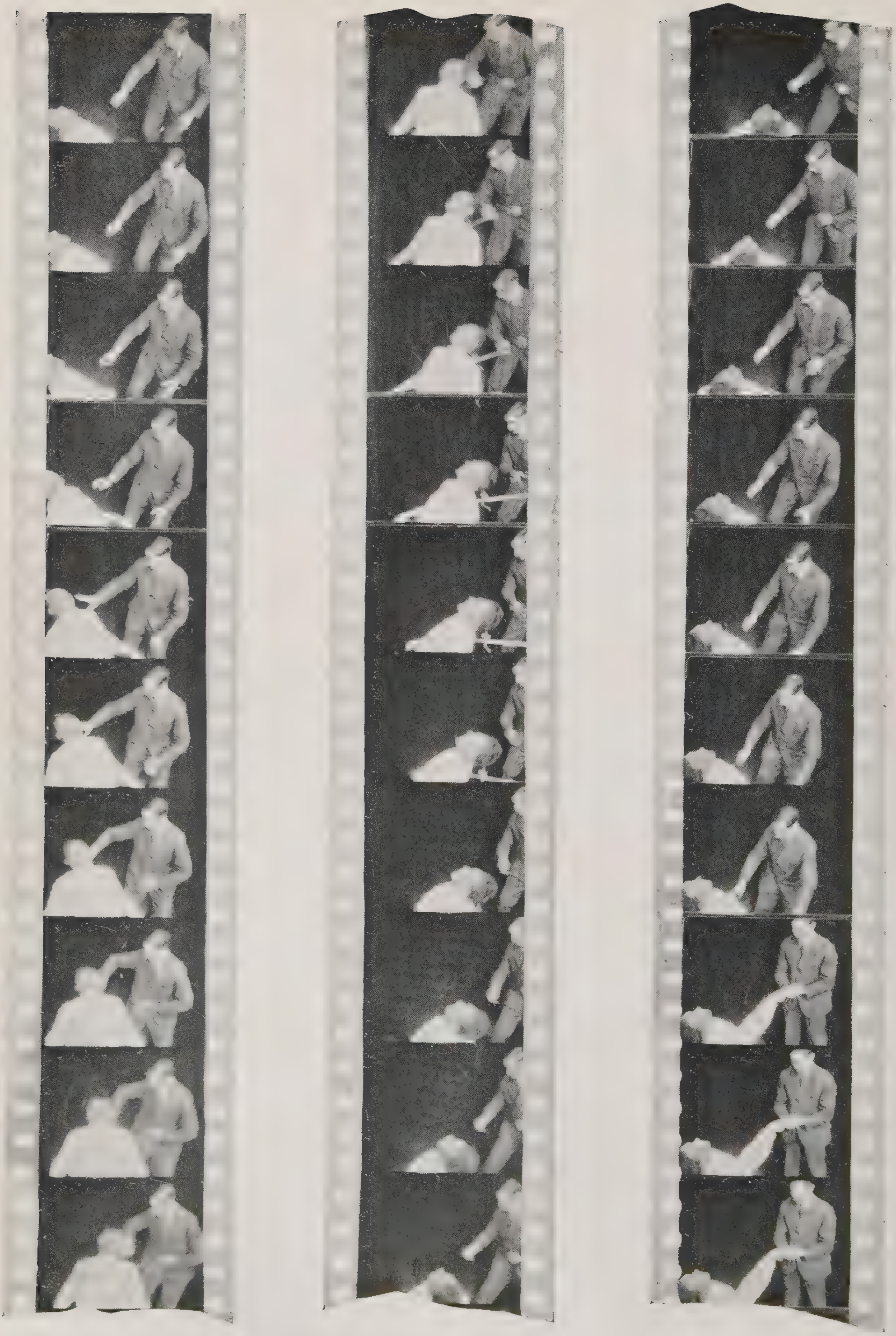

Fig. 68.—Turning [illegible] the right, head back 60°.

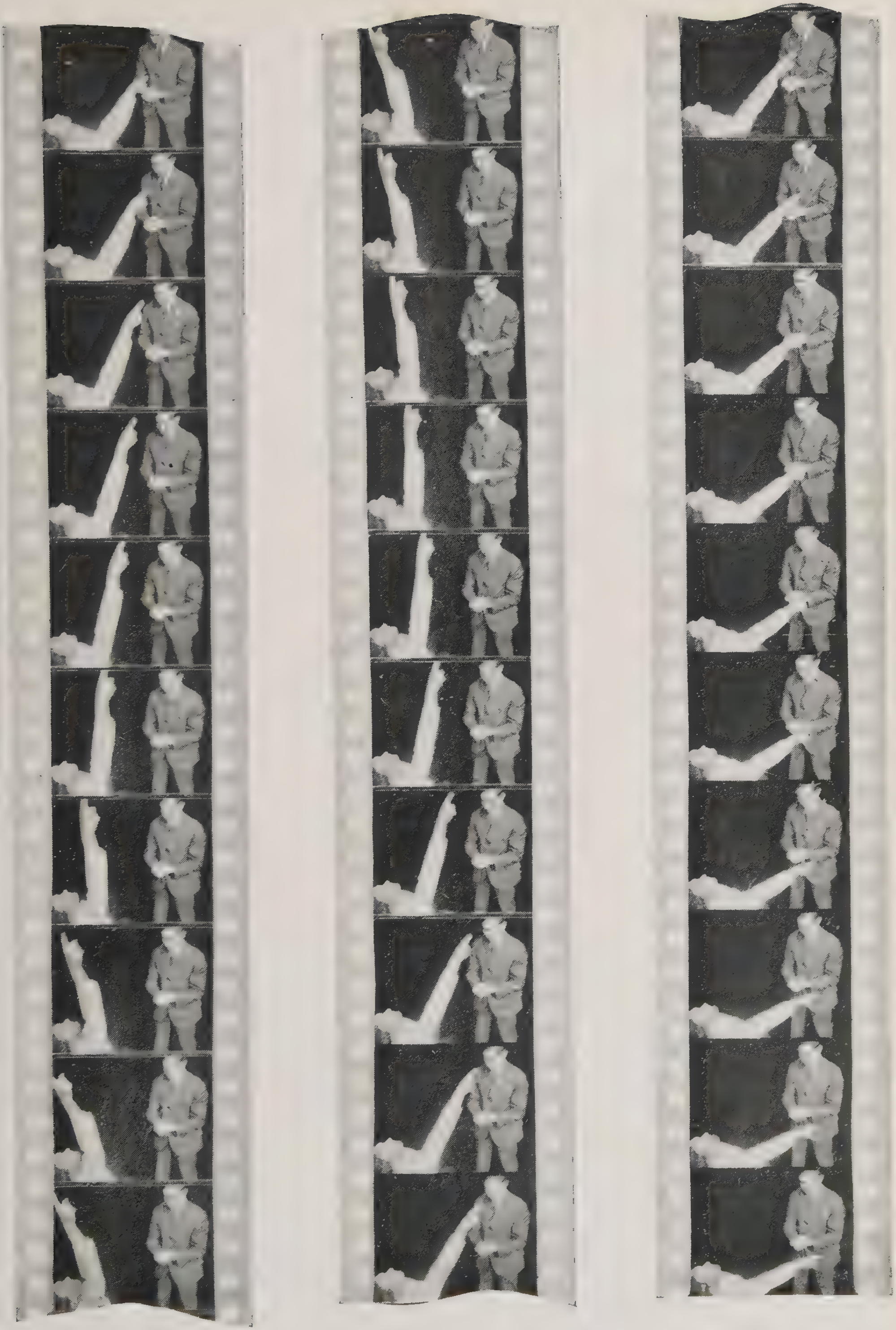

FIG. 69.—Produces past-pointing to the right.

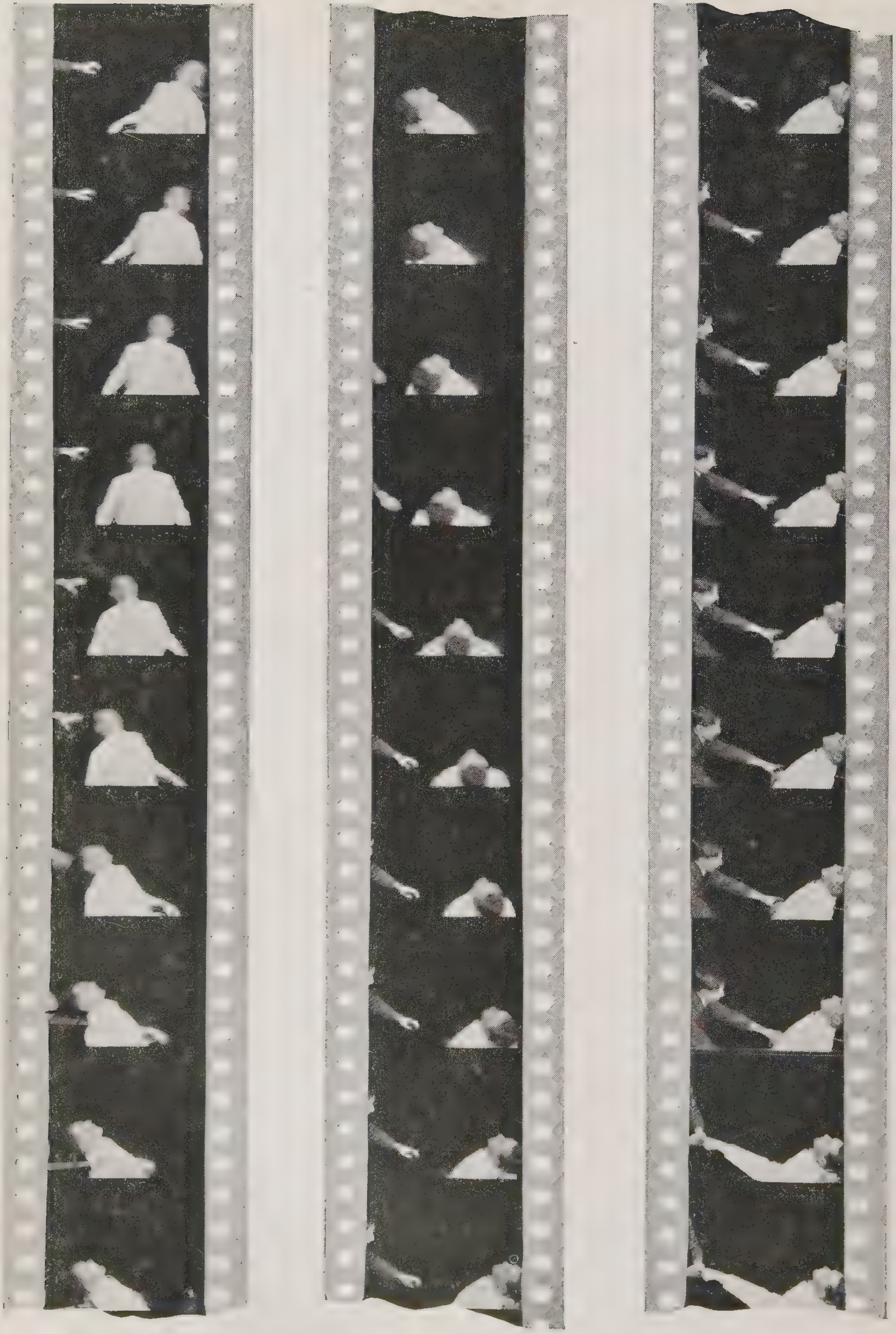

FIG. 70.—Turning to the left, head 60° back, causes endolymph movement to the left.

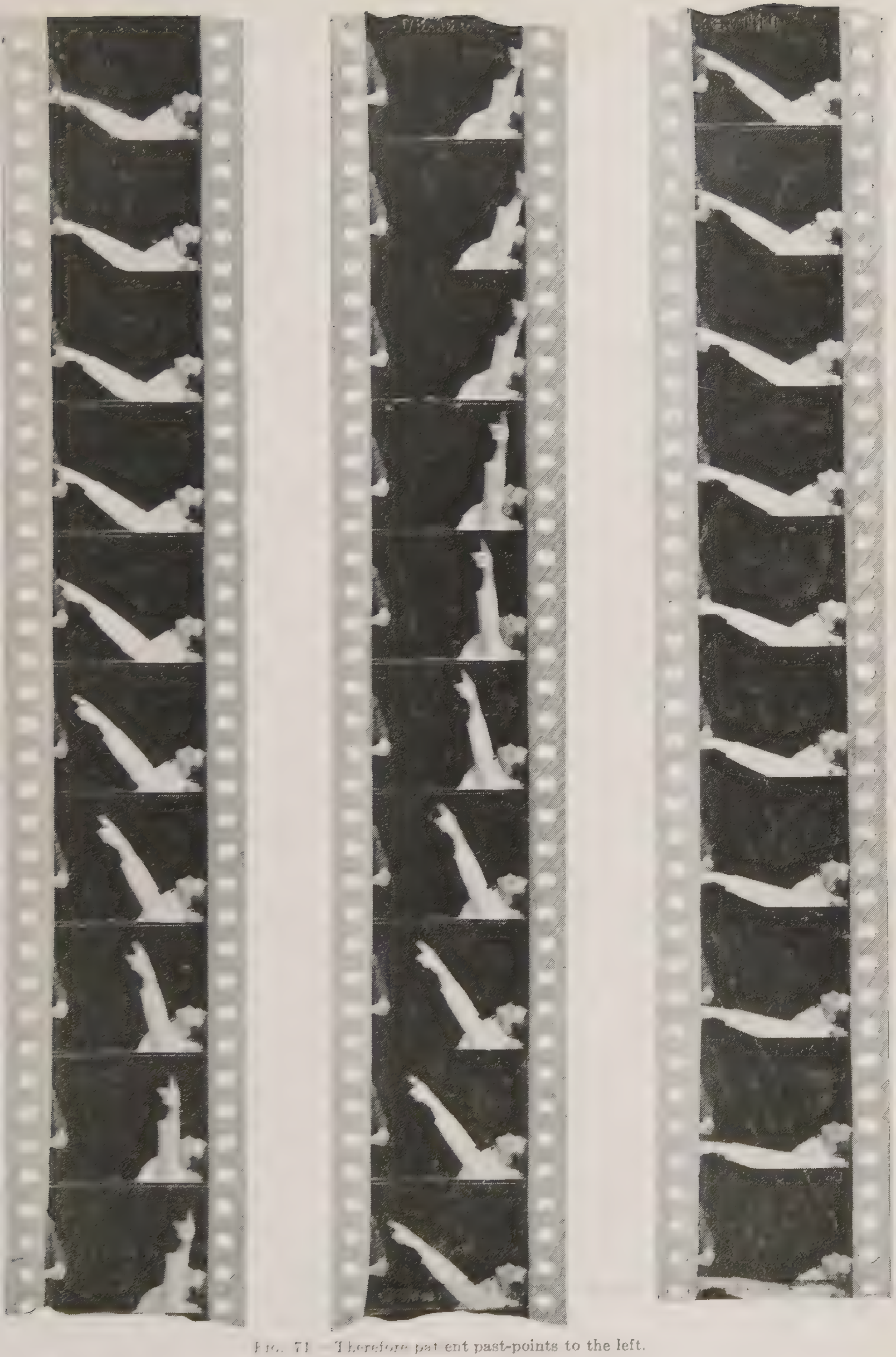

FIG. 71.—Therefore patient past-points to the left.

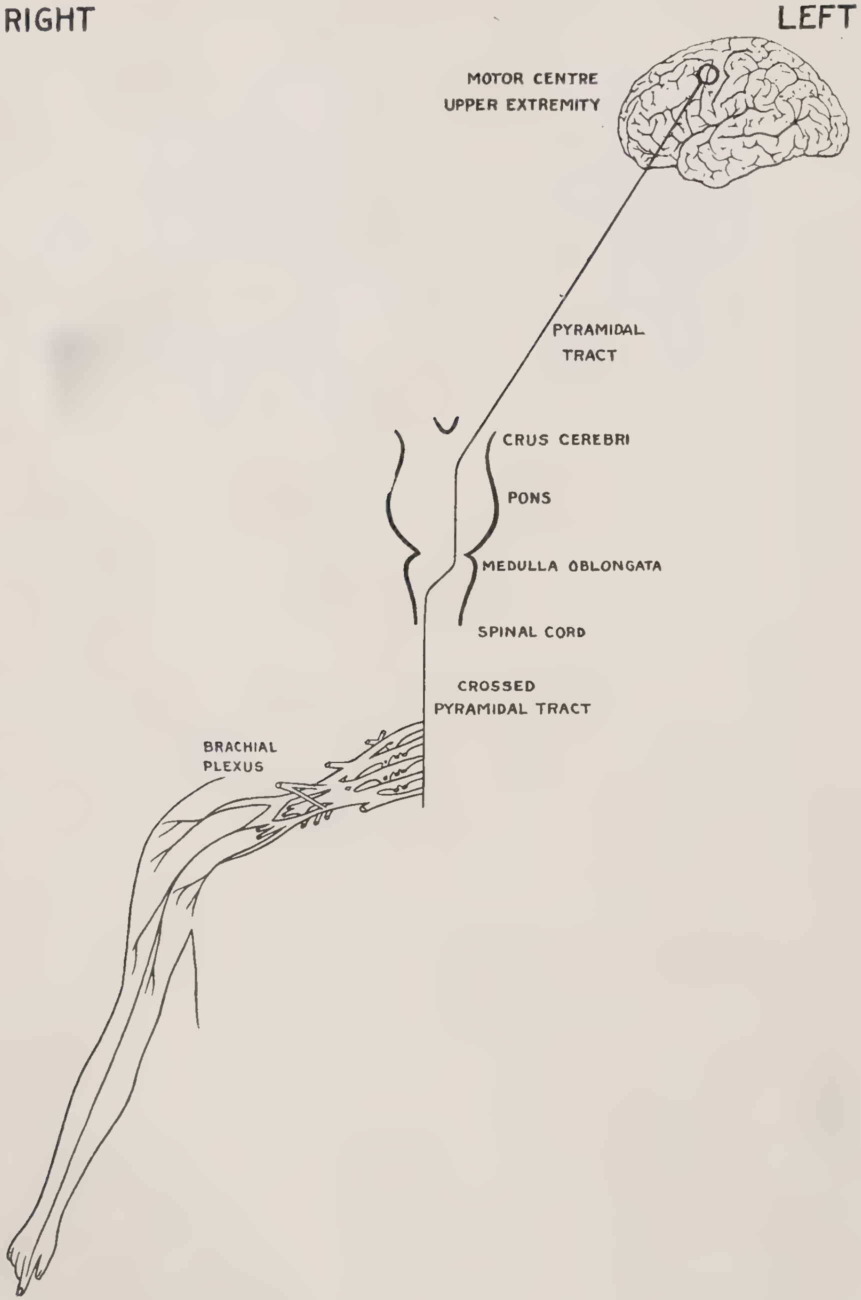

Fig. 72.—Pyramidal tract becoming the crossed pyramidal tract, ("power" tract).

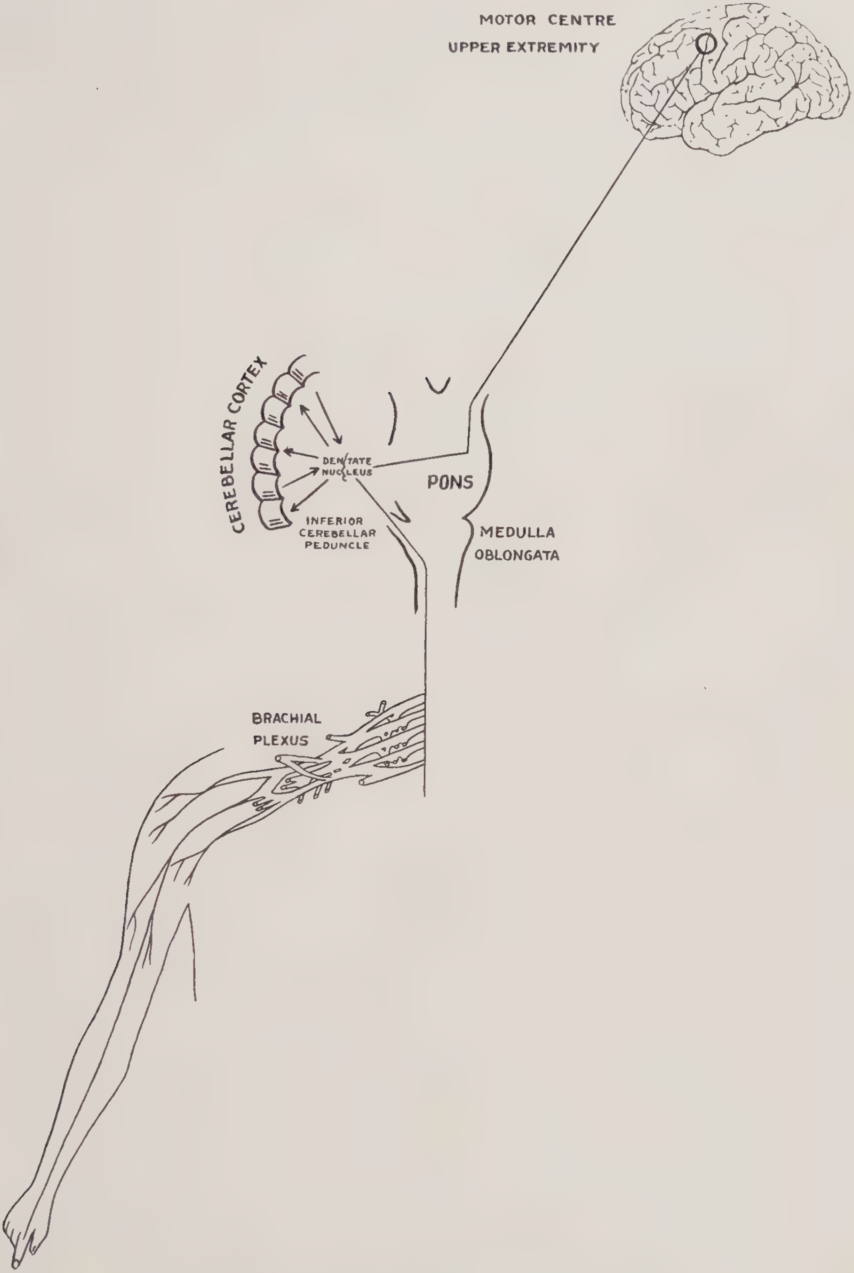

Fig. 73.—Cerebro-cerebello-spinal tract, ("accuracy" tract).

In the previous chapter were shown the paths of the sensory impulses from the ear to the cerebral cortex producing vertigo. The paths for pointing, on the other hand, are purely motor. They begin in the motor area of the cerebral cortex and end in the peripheral distribution to those particular muscles which are employed in the pointing tests. These paths are two:

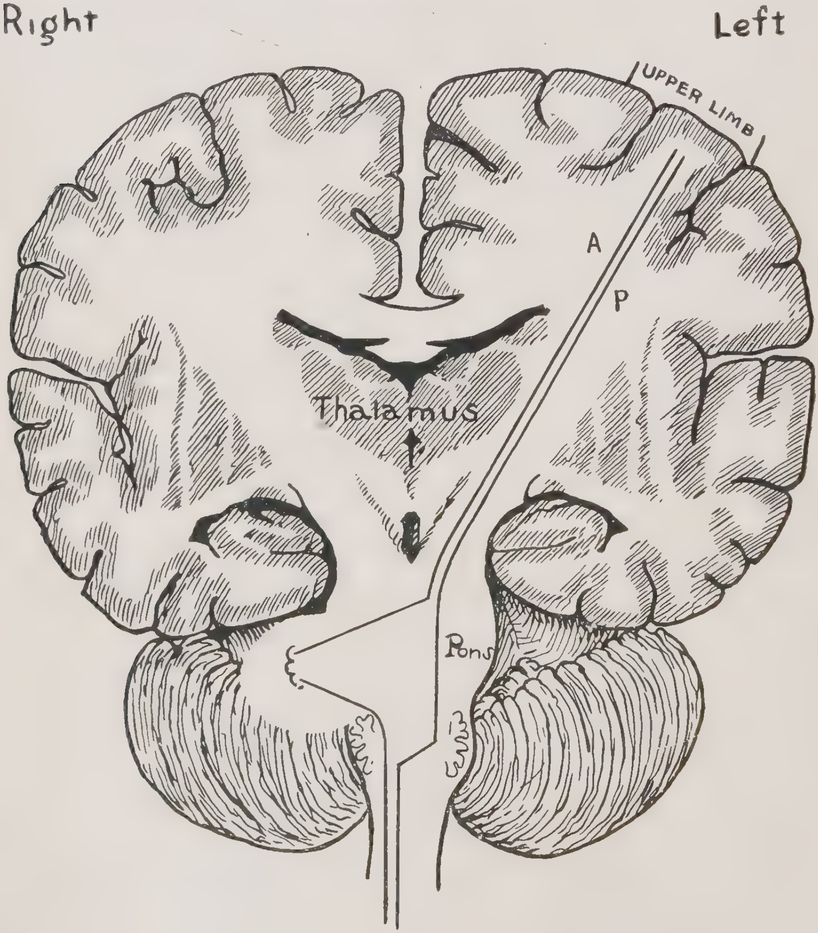

Fig. 74.—Pointing tracts: "P" "Power tract," pyramidal tract, decussating in medulla oblongata. "A" "Accuracy tract," cerebro-cerebello-spinal tract, decussating in pons.

(1) The pyramidal tract. This may be spoken of as the *power* tract. It supplies the motor force for pointing.

(2) The cerebro-cerebello-spinal tract. This may be termed the *accuracy* tract. It is by this path that the cerebellum is able to exercise its synergic or inhibitory influence. The pyramidal tract alone would be able to supply the necessary energy to raise the arm up and to bring the arm down. But it is the cerebellum that enables the arm to point accurately and *find the finger.* Just how this is accomplished may be explained in the following way: In the cerebellar cortex are separate centres, one for the outward pointing and one for the inward pointing of different joints. As the arm descends to find the finger, if it tends to point outward, the inward-pointing centre restrains this aberrant movement and draws it back to its proper course. If it tends to point inward, the outward-pointing centre of the cerebellum brings it back to its proper course.

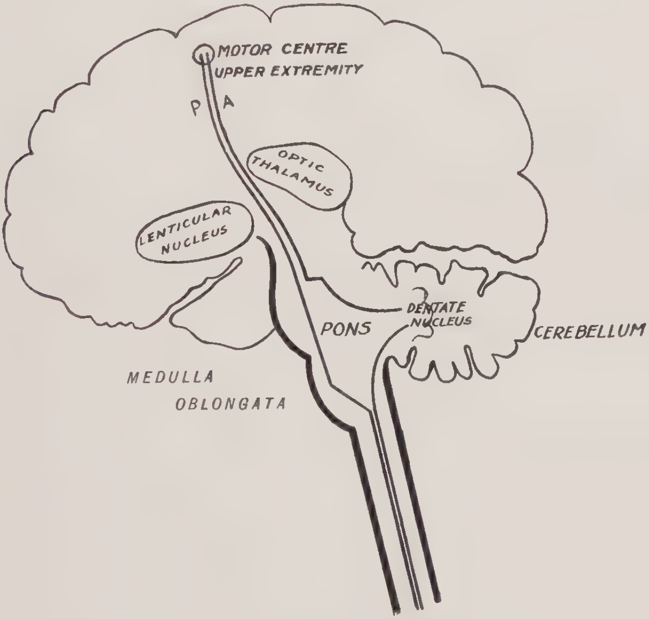

FIG. 75.—Pointing tracts. "P" "Power tract"—pyramidal tract, decussating in medulla oblongata. "A" "Accuracy tract"—cerebro-cerebello-spinal tract, decussating in pons.

Consulting the diagram (Fig. 72) the pyramidal tract for the pointing of the right upper extremity starts in the left cerebral cortex at the motor centre of the upper extremity. Traversing the internal capsule it passes through the left cerebral crus, the left side of the pons, and decussates in the medulla oblongata to the right side, becoming the crossed pyramidal tract. Passing down the right side of the spinal cord the fibres enter the brachial plexus and are distributed to the muscles of the right upper extremity.

The cerebro-cerebello-spinal tract (Fig. 73), for the pointing of the right upper extremity, also begins in the left cerebral cortex in the motor centre of the upper extremity. Traversing the internal capsule it passes through the left crus cerebri into the left side of the pons where it probably decussates to the opposite side and enters the dentate nucleus of the cerebellum through the right middle cerebellar peduncle. The dentate nucleus is a relay station for fibres to and from the cerebellar cortex. Continuing from the dentate nucleus the pathway passes through the right inferior cerebellar peduncle through the right side of the medulla oblongata and spinal cord to enter the brachial plexus through which it also is distributed to the muscles of the right upper extremity.

Figure 74 combines the two pathways in one diagram.

Figure 75 represents a lateral view of the two pathways; the pyramidal or ''power'' tract is shown decussating in the medulla oblongata and the cerebro-cerebello-spinal or ''accuracy'' tract is shown decussating in the pons.

## Planes of Past-pointing

Past-pointing can occur in three planes of space—the horizontal, the frontal and the sagittal. The plane of the past-pointing is always in the plane of the vertigo producing it; the direction of the past-pointing is naturally directly opposite to the direction of the vertigo producing it. After turning an individual with the head upright, the horizontal canals produce a sensation of turning in the horizontal plane. Past-pointing, therefore, occurs in the horizontal plane to the right or to the left in the direction opposite to the subjective sensation of turning. For instance, after turning to the right the patient stops, feels that he is moving to the left away from the finger, and therefore past-points to the right.

Now if the head is inclined 120° forward, the turning sensation is in the frontal plane of the head. If the head is kept in this position, the frontal plane is parallel to the plane of the floor. Therefore the sensation of turning is the same as when he was turned with the head upright. If he has been turned to the right he feels that he is turning to the left and therefore past-points to the right. Now if after turning the head is raised, the frontal plane then assumes a position at a right angle to the floor. The subjective sensation therefore is one of *falling* to the left in the frontal plane. As he feels that he is falling to the left he past-points to the right. It will be noted that the result is the same—after turning with the head forward, he will past-point to the right whether the head is kept in the forward position or brought upright after the turning.

Now if the individual has been turned with the head inclined backward 60°, on stopping, the turning sensation is also in the frontal plane. If the head is kept in this position, the frontal plane of the head is parallel to the floor. Therefore the sensation of turning is the same as when he was turned with the head upright. If he has been turned to the right he feels, therefore, that he is going to the left and consequently he past-points to the right. If, however, after turning to the right with the head back the head is brought upright, the frontal plane assumes a position at a right angle to the floor. Immediately the previous sensation of turning to the left is changed into a sensation of falling to the right. As the individual feels he is falling to the right, he past-points to the left. It will be noted that bringing the head upright after turning with the head back causes a complete reversal of the past-pointing; if the head is kept back, he past-points to the right, whereas if the head is brought upright he past-points to the left. The explanation is simple and strictly according to rule. The past-pointing is in the direction opposite to the vertigo. We always consider vertigo in terms of our relation to what is in front of us, or below us. After the individual has been turned to the right with the

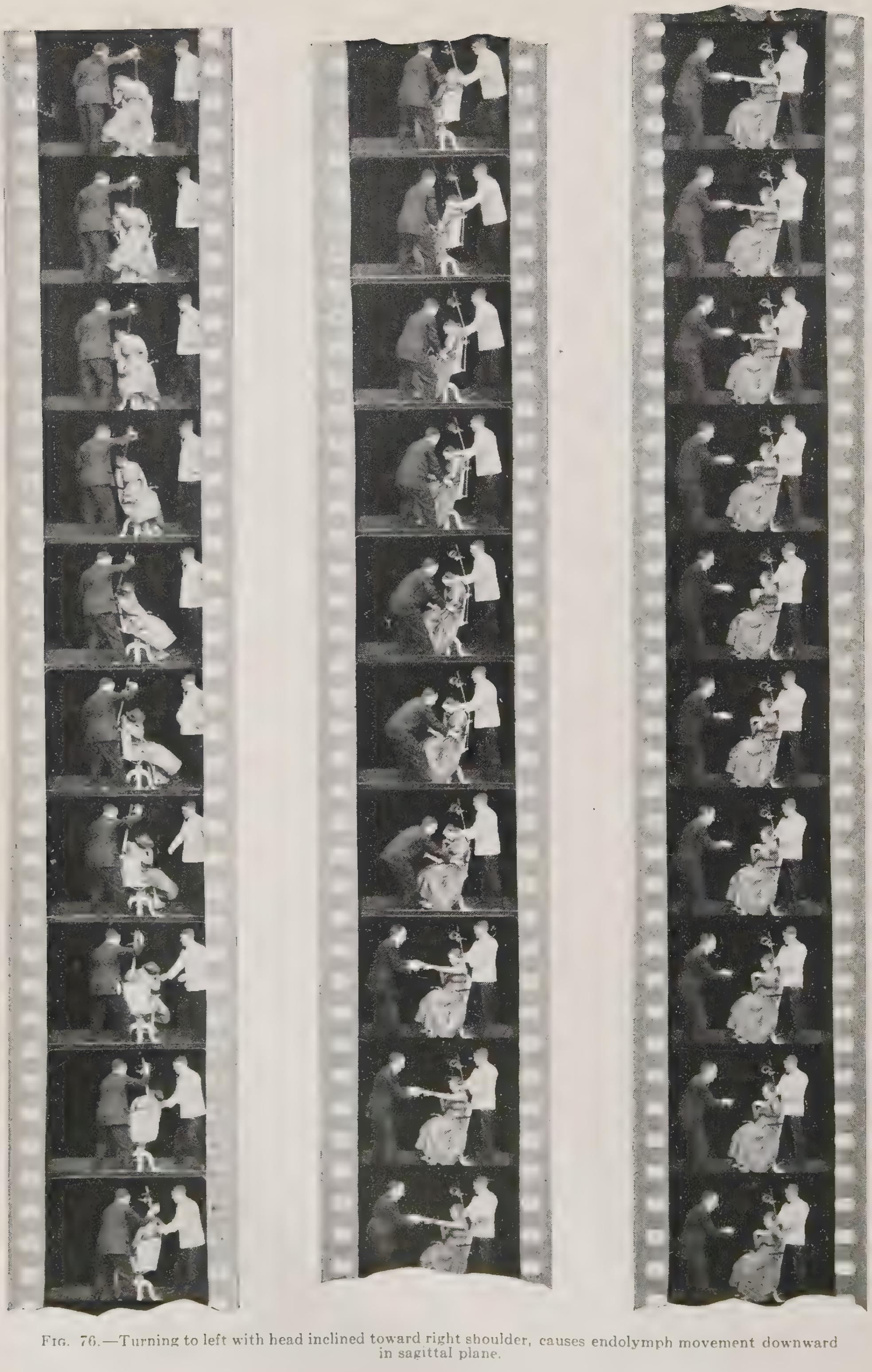

FIG. 76.—Turning to left with head inclined toward right shoulder, causes endolymph movement downward in sagittal plane.

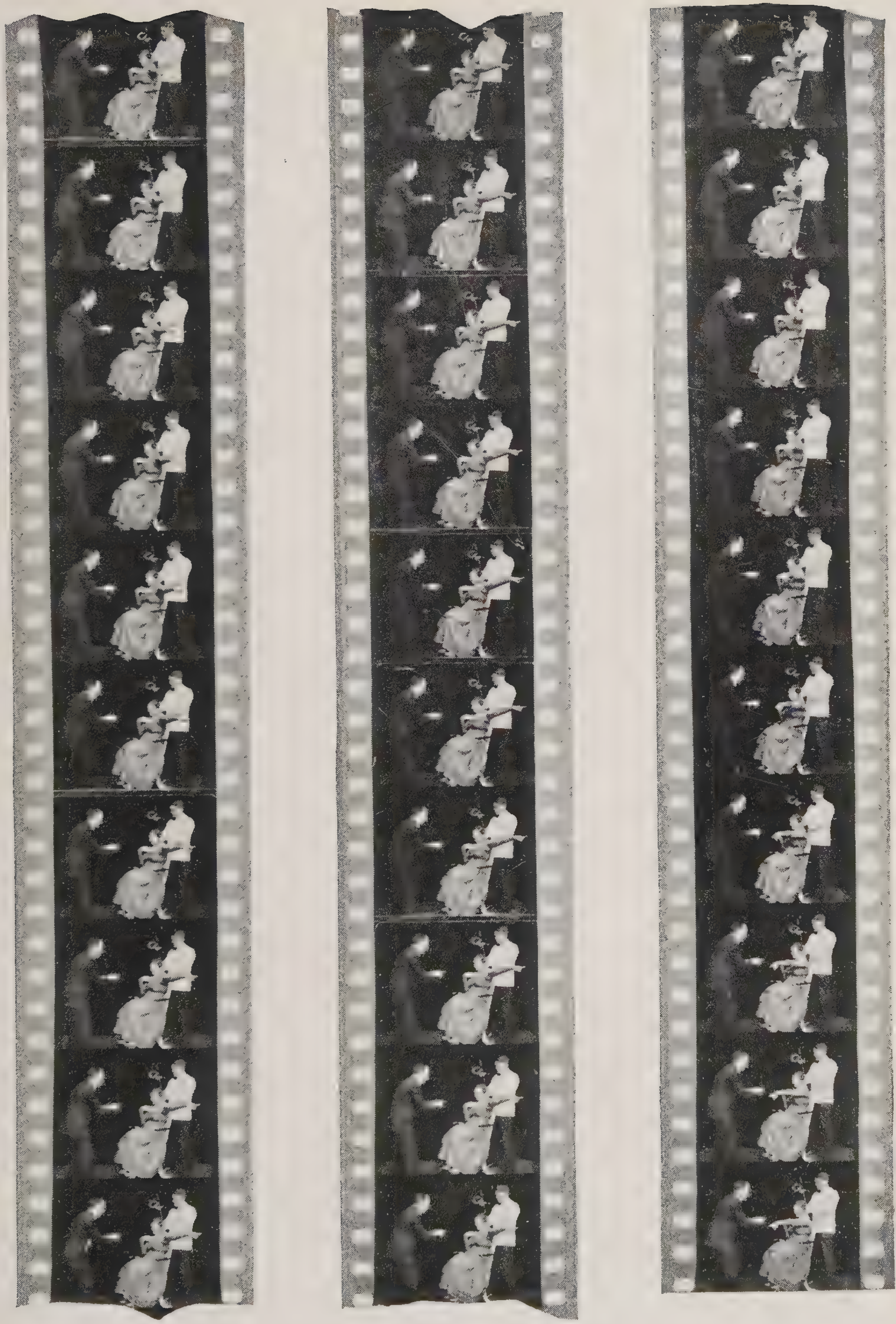

FIG. 77.—Therefore patient past-points below

head back, the endolymph movement is to the *right* in relation to objects in front of him. When, however, the head is brought upright, this same endolymph movement is to the *left* in relation to objects above him. His sensation of vertigo is therefore immediately reversed when the head is brought upright. Anyone tested in this way at once recognizes this reversal in the vertigo.

Now if the head is inclined toward the shoulder, turning produces the subjective sensation of movement in the sagittal plane of the head. If the head is maintained in this position, the sagittal plane of the head is parallel to the floor; the individual therefore past-points in the horizontal plane, to the right or the left. If, however, the head is brought upright, the sagittal plane of the head is then at right angles to the floor; the individual therefore has a subjective sensation of falling in the sagittal plane—forward or backward. Consequently he past-points *below* or *above* the finger of the examiner, in the sagittal plane.

Figures 76 and 77 show turning to the left with the head inclined to the right shoulder. The plane of the turning is therefore in the sagittal plane of the head. At the end of the turning the patient raises her head to the vertical position, the examiner has the patient feel his finger, move the arm out to the side; on returning to the median line the patient is unable to find the examiner's finger but past-points below it.

## TABLE OF PAST-POINTING AFTER TURNING

Turning to the right with the head upright produces past-pointing to the right.

Turning to the left with the head upright produces past-pointing to the left.

Turning to the right with the head back 60° produces past-pointing to the right.

Turning to the left with the head back 60° produces past-pointing to the left.

Turning to the right with the head forward 120° produces past-pointing to the right.

Turning to the left with the head forward 120° produces past-pointing to the left.

Turning to the right with the head inclined toward the right shoulder and then bringing head upright produces past-pointing upward.

Turning to the left with the head inclined toward the right shoulder and then bringing head upright, produces past-pointing downward.

Turning to the right with the head inclined toward the left shoulder and then bringing head upright, produces a past-pointing downward.

Turning to the left with the head inclined toward the left shoulder and then bringing head upright, produces a past-pointing upward.

## Table of Past-pointing After Douching

Douching the right ear with the head upright, water 68°, produces past-pointing to the right.

Douching the left ear with the head upright, water 68°, produces past-pointing to the left.

Douching the right ear, head back 60°, water 68°, produces past-pointing to the right.

Douching the left ear, head back 60°, water 68°, produces past-pointing to the left.

Douching the right ear with the head forward 120°, water 68°, produces past-pointing to the left.

Douching the left ear with the head forward 120°, water 68°, produces past-pointing to the right.

Douching the right ear with the head upright, water 112°, produces past-pointing to the left.

Douching the left ear with the head upright, water 112°, produces past-pointing to the right.

Douching the right ear, head back 60°, water 112°, produces past-pointing to the left.

Douching the left ear, head back 60°, water 112°, produces past-pointing to the right.

Douching the right ear, head forward 120°, water 112°, produces past-pointing to the right.

Douching the left ear, head forward 120°, water 112°, produces past-pointing to the left.

## Falling

It may be noted at this point that the phenomenon of "falling" after ear-stimulation may be regarded as a past-pointing of the entire body. The patient falls because of the vertigo, and not because his body is drawn to one side or the other by the ear-stimulation. Falling is naturally not in evidence when the subjective sensation is one of movement in the horizontal plane; the individual feels that he is being rotated on his own vertical axis and there is therefore no tendency to fall either to the right or to the

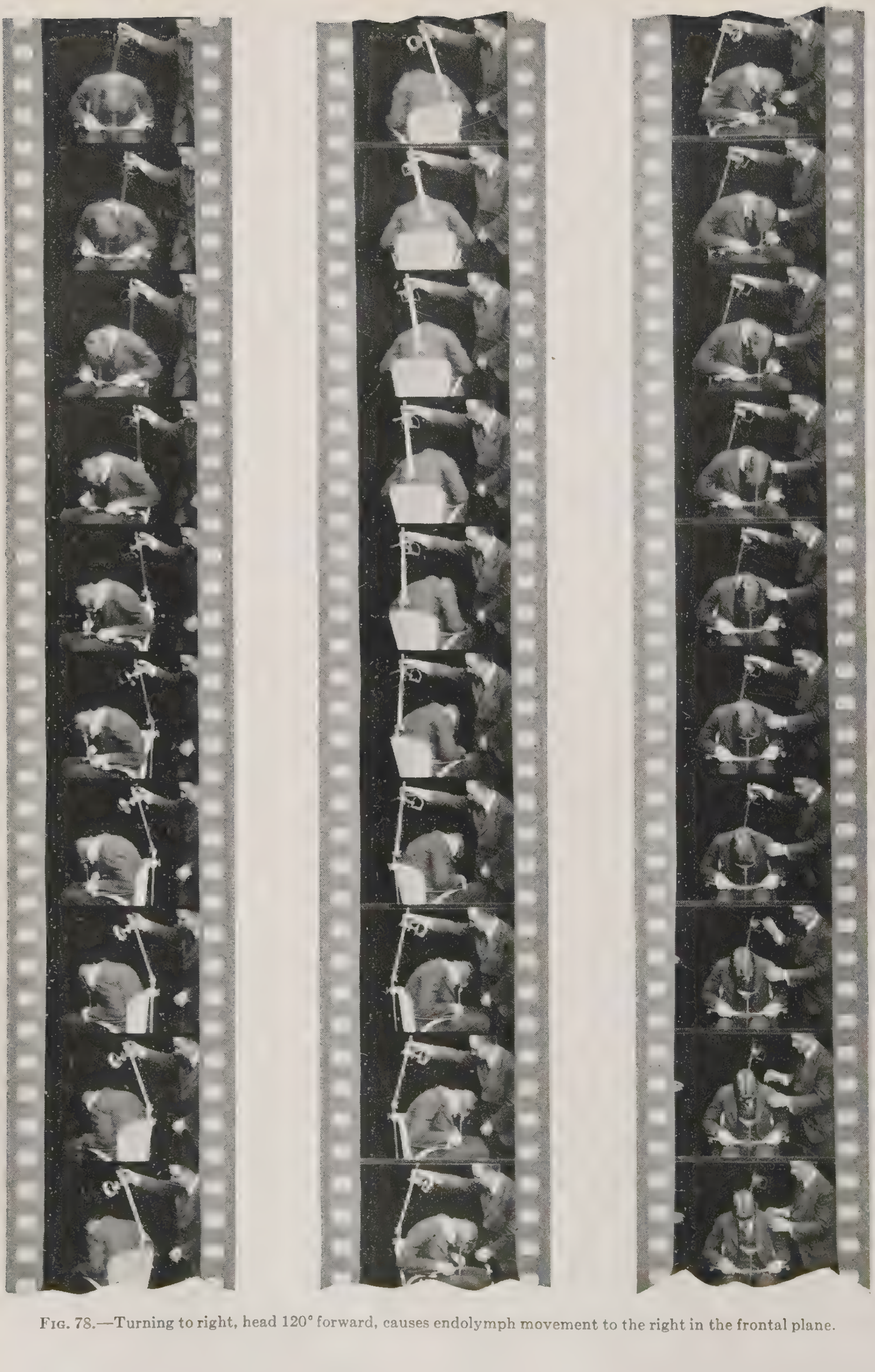

FIG. 78.—Turning to right, head 120° forward, causes endolymph movement to the right in the frontal plane.

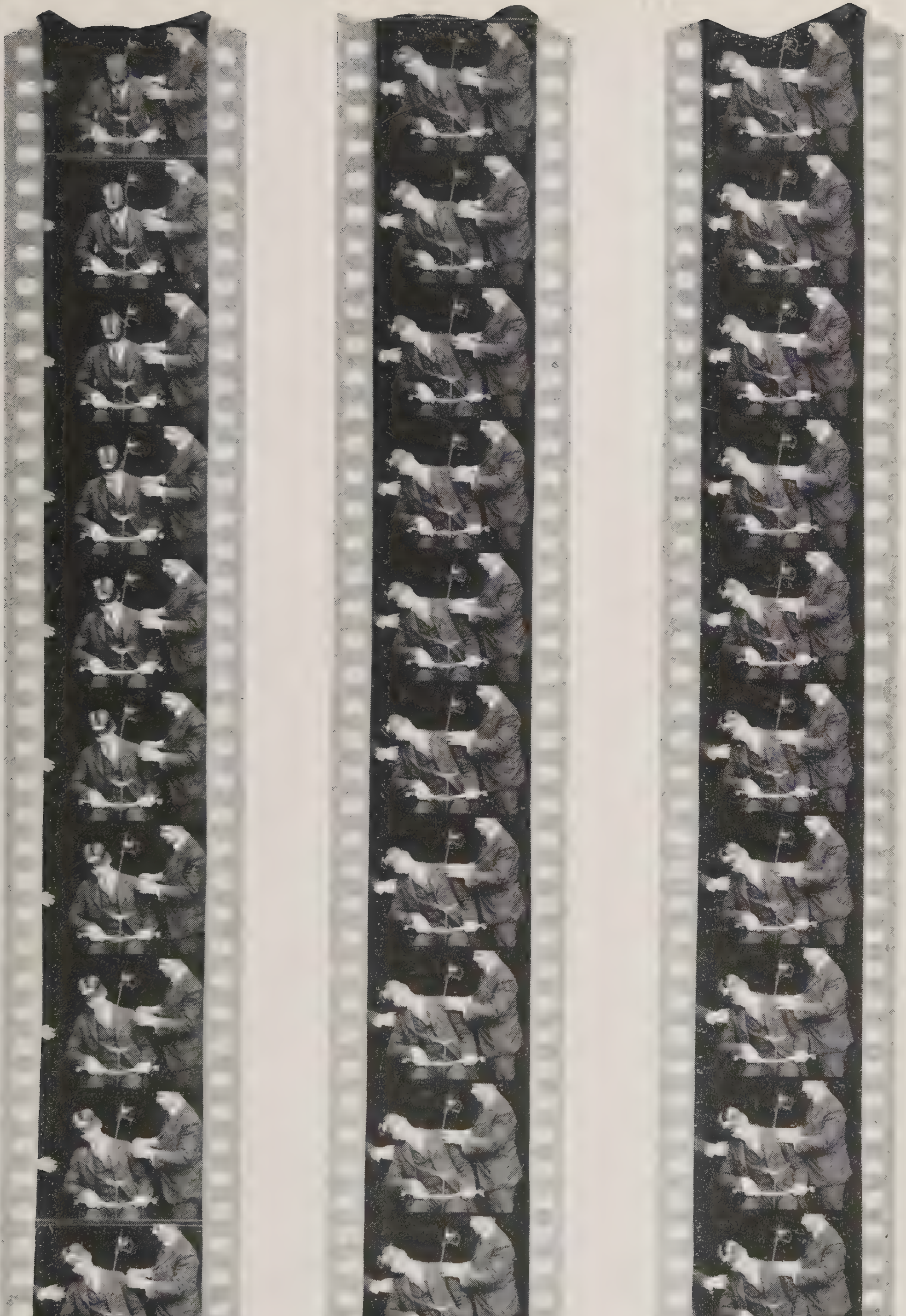

FIG. 79.—Therefore on sitting up, patient falls to the right in the frontal plane

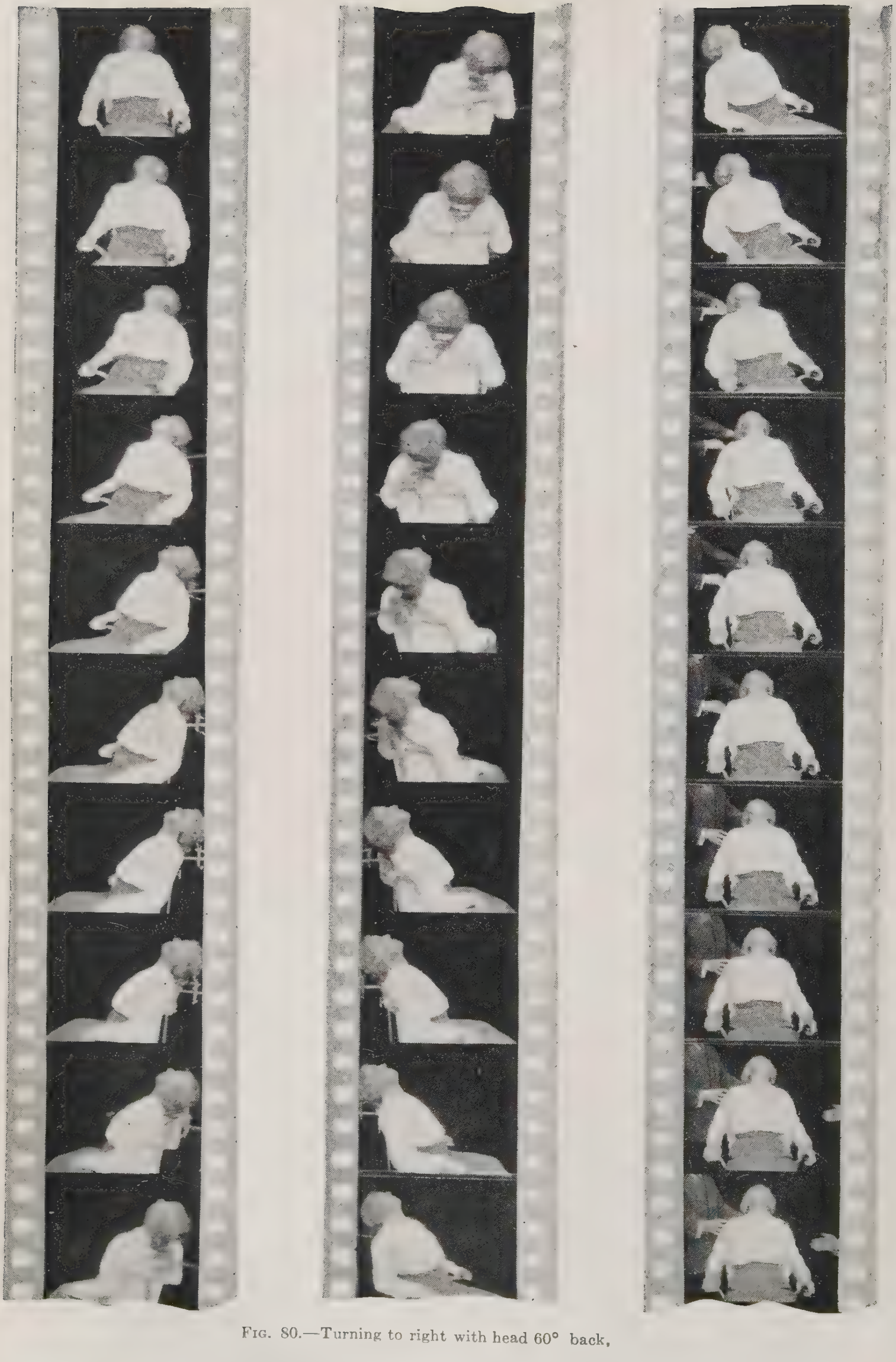

FIG. 80.—Turning to right with head 60° back,

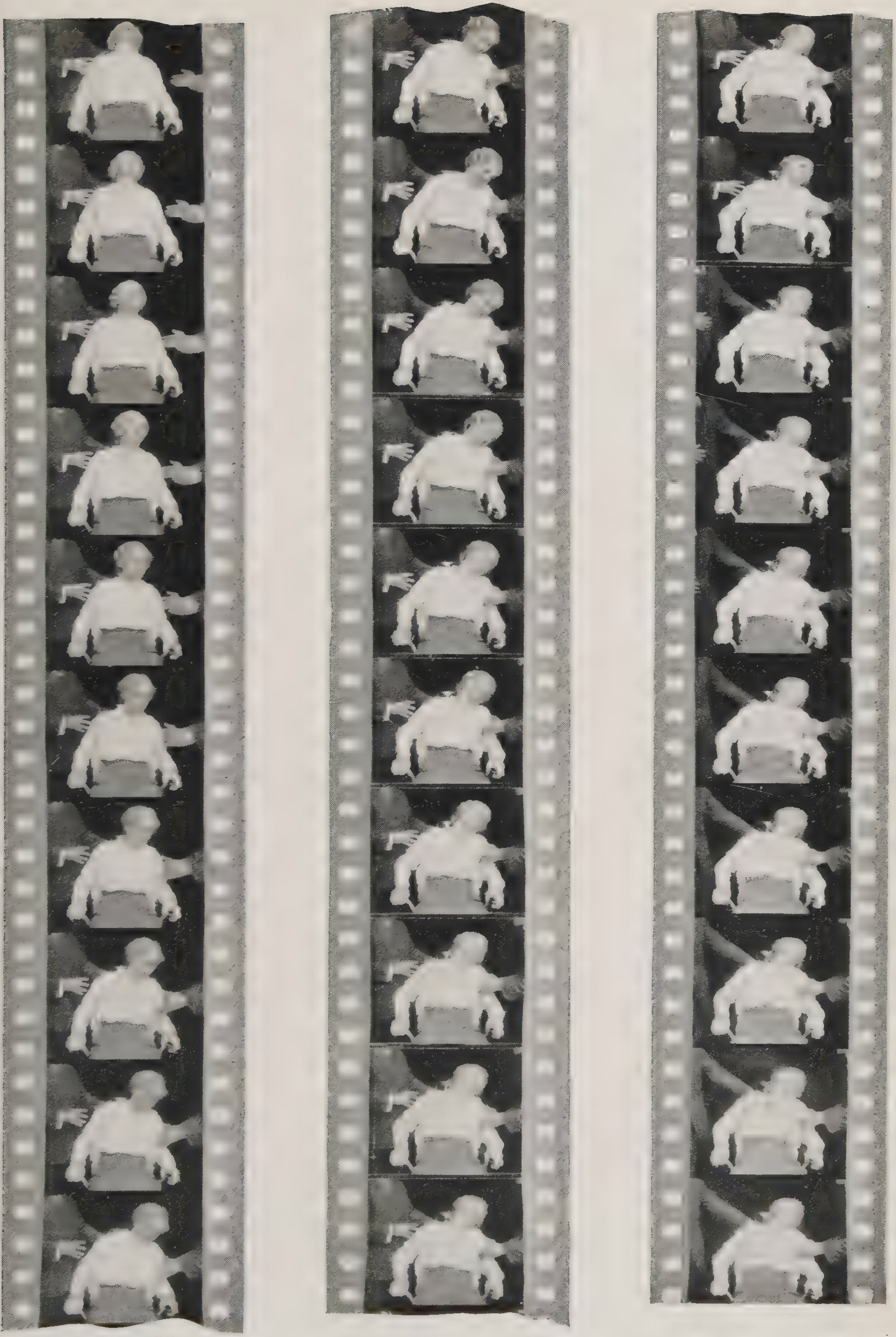

FIG. 81.—Will produce falling to left when head is raised to upright position.

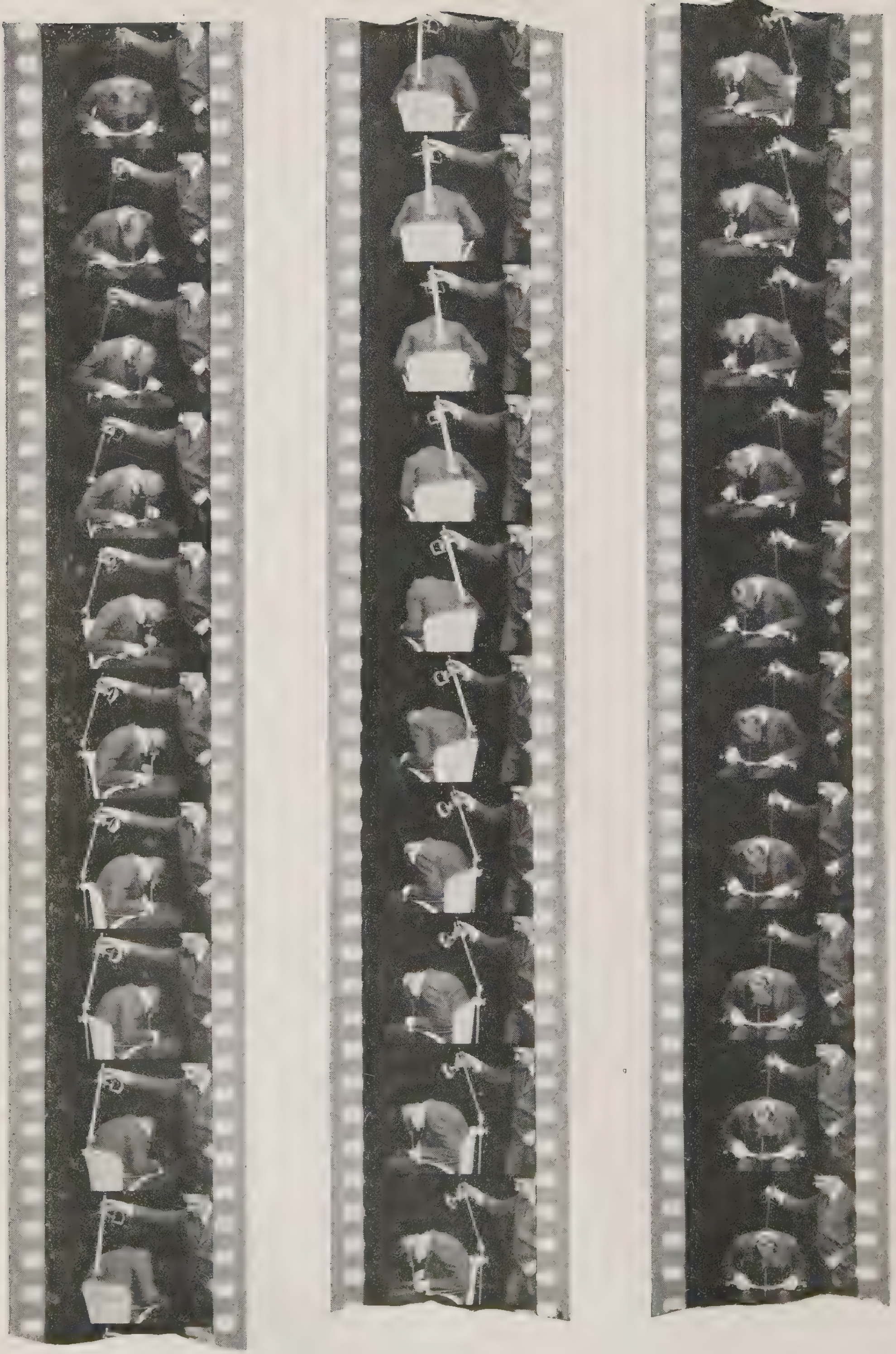

Fig. 82.—Turning to left, head 120° forward, causes endolymph movement to the left in the frontal plane.

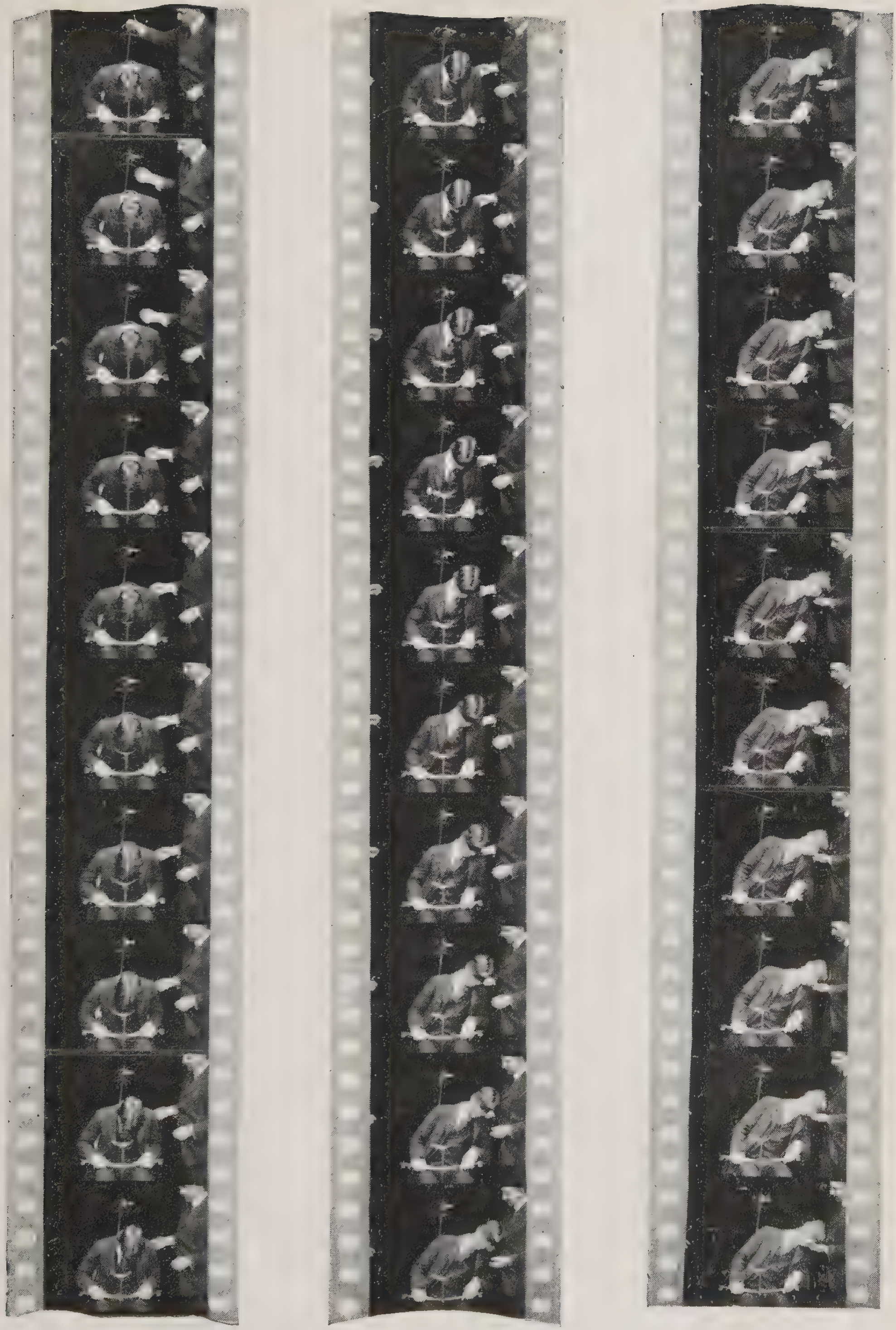

FIG. 83.—Therefore on sitting up, patient falls to the left in the frontal plane.

left or front or back. If, however, the subjective sensation is one of turning in the frontal plane, he tends to fall either to one side or the other. If the subjective sensation is one of movement in the sagittal plane, he tends to fall either forward or backward.

The pictures (Figs. 78, 79) show the patient being turned to the right with the head forward; after the chair is stopped the patient raises his head to the vertical position and falls directly to the right. When the head is in the upright position, the endolymph movement is to the right and therefore the falling is to the right. In detail, endolymph movement to the right causes a subjective sensation of falling to the left, and therefore the patient falls to the right.

The pictures (Figs. 80, 81) show the patient being turned to the right with the head *back;* after the chair is stopped the patient brings the head forward to the vertical position and falls directly to the left. With the head in the upright position the endolymph movement is to the left and the patient therefore falls to the left. In detail, the endolymph movement to the left causes a subjective sensation of falling to the right and therefore the patient falls to the left.

The pictures (Figs. 82, 83) show the patient being turned to the left with the head forward; after the chair is stopped the patient raises the head to the upright position and immediately falls to the left, because the endolymph movement is to the left.

All of these falling movements to the right and to the left have occurred in the frontal plane because turning with the head forward or backward stimulates the vertical canals in the frontal plane. Falling in the *sagittal* plane is necessarily produced by stimulation of the vertical canals in the sagittal plane.

The pictures (Fig. 84) show the patient being turned to the left with the head inclined toward the right shoulder. The plane of the turning is therefore in the sagittal plane of the head. When the chair is stopped, the patient brings the head to the upright position and immediately falls forward in the sagittal plane.

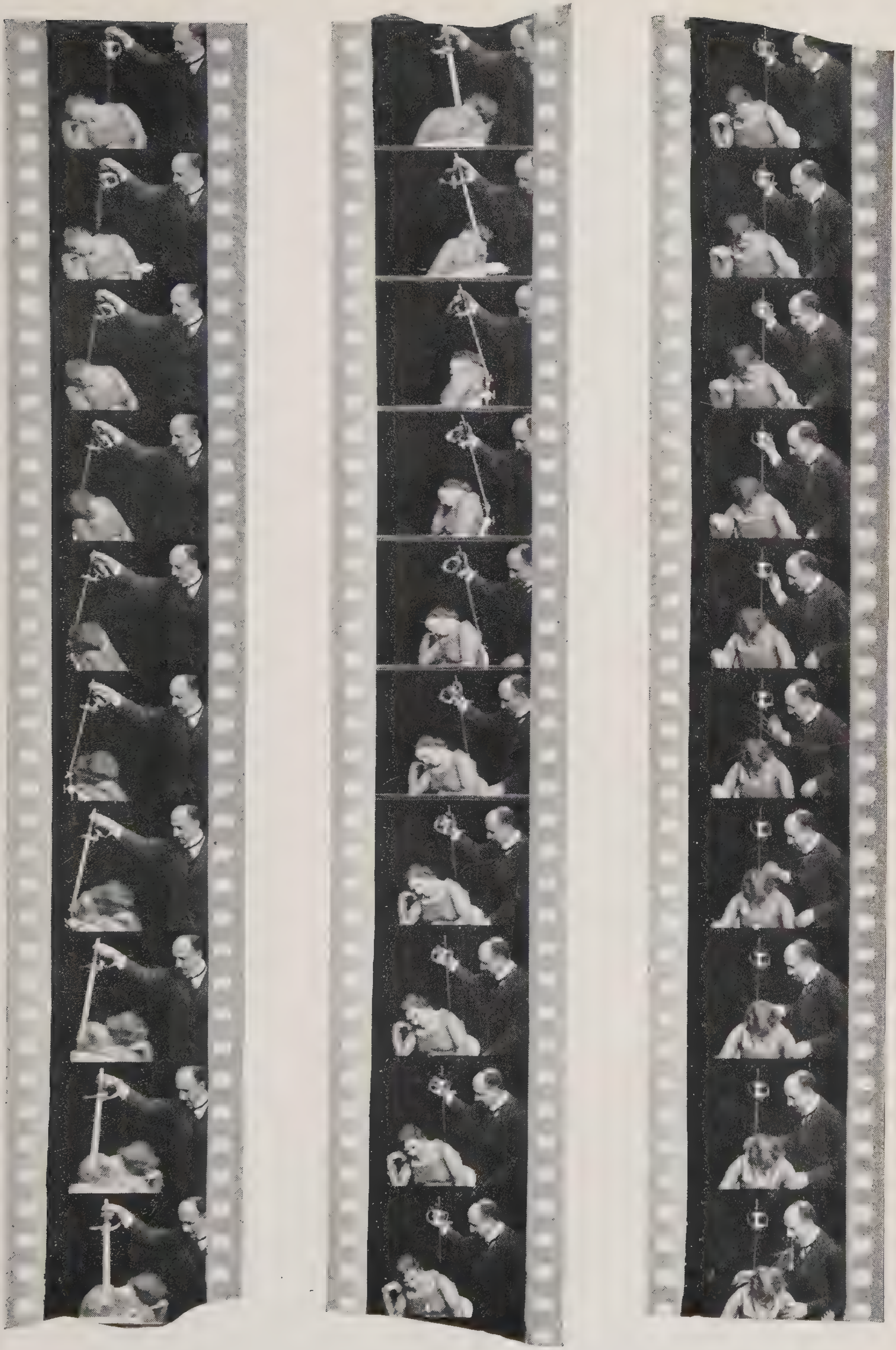

FIG. 84.—Turning to left, head inclined toward right shoulder, causes endolymph movement downward in sagittal plane. Therefore patient falls forward.

## TABLE OF FALLING AFTER TURNING

Turning to the right, head forward 120°, and sitting upright, produces falling to the right.

Turning to the left, head forward 120°, and sitting upright, produces falling to the left.

Turning to the right, head back 60°, and sitting upright, produces falling to the left.

Turning to the left, head back 60°, and sitting upright, produces falling to the right.

Turning to the right, head inclined toward the right shoulder, and bringing head upright, produces falling backward.

Turning to the left, head inclined toward the right shoulder, and bringing head upright, produces falling forward.

Turning to the right, head inclined toward the left shoulder, and bringing head upright, produces falling forward.

Turning to the left, head inclined toward the left shoulder, and bringing the head upright, produces falling backward.

## TABLE OF FALLING AFTER DOUCHING

Douching right ear, head upright, water 68°, produces falling to the right.
Douching left ear, head upright, water 68°, produces falling to the left.
Douching right ear, head back 60°, water 68°, produces falling to the right.
Douching the left ear, head back 60°, water 68°, produces falling to the left.
Douching right ear, head upright, water 112°, produces falling to the left.
Douching left ear, head upright, water 112°, produces falling to the right.
Douching right ear, head back 60°, water 112°, produces falling to the left.
Douching left ear, head back 60°, water 112°, produces falling to the right.

It is not necessary to commit to memory any one of these responses. Under all circumstances both the *past-pointing and the falling are always in the direction of the endolymph movement.*

# CHAPTER XVIII

## TECHNIC OF EXAMINATION OF THE AUDITORY APPARATUS AND OF THE VESTIBULAR APPARATUS

In the last three chapters we have discussed nystagmus, vertigo, and the pointing-tests as such; we are therefore in a position to understand the object of the examinations and can now take up the technic. So little has been known of the physiology of the vestibular labyrinth until comparatively recent times that the lack of a well-established technic of its examination is not surprising. Until recently, all examinations of the labyrinth were carried out with the sole purpose of determining its own integrity. Such tests were sufficient for the aural surgeon, who was called upon merely to decide whether or not to operate on the ''end-organ.'' With the development of the idea that the labyrinth is only one portion of the vestibular apparatus came the realization that this whole apparatus was being interrogated at the same time that the internal ear itself was being tested, and also that an intelligent interpretation of the phenomena obtained by such tests can give the examiner an insight into the condition of those various brain paths and brain centres in relation with the internal ear. Thus, when a known stimulus is applied to the labyrinth, any response obtained therefrom, be it nystagmus or vertigo, indicates not only a functionating and reacting labyrinth, but also intact pathways from the labyrinth to the brain centres responsible for those reactions. Conversely, the non-appearance of any of the normal responses to stimulation indicates an interruption at some point along the particular pathway that fails to produce that particular response. This made it evident that the old technic of examination was inadequate and Barany elaborated a new technic which created a standard for these examinations. In preceding chapters we have shown that the fibres from the horizontal semicircular canal have an entirely separate intracranial course from those of the vertical semicircular canals; and while all fibres concerned in vertigo go to the cerebellum, we consider that those from the horizontal canal reach it by way of the inferior cerebellar peduncle, while those from the vertical canals go by way of the middle cerebellar peduncle. Further, we have shown that nystagmus and vertigo are distinct and separate phenomena; also that past-pointing is not a "cerebellar pull," but is a cerebral phenomenon exclusively and depends entirely on the vertigo induced by the ear stimulation. Barany's technic, elaborated before such a differentiation was demonstrated, deals with the labyrinth as a whole, whereas from this new viewpoint, it at once becomes evident how important it is to examine *each set of canals separately* and furthermore to analyze separately the nystagmus, vertigo and past-pointing produced by stimulation of each set of canals. Above all, it is of prime importance that if the data obtained from such an examination are to be relied on, the technic must be accurate, complete and painstaking. Those unconvinced of the value of these tests must ascribe their difficulty to laxity in the methods of conducting them. An examination of the labyrinth, although the underlying principles are by no means complex, nevertheless requires the determining of so much data that it is an almost hopeless task when examining a patient to attempt to write down all the findings in *words*. The beginner especially is bewildered by the apparently endless amount of data. All who have done this work realize what a nuisance it is to keep haphazard records, on slips of paper perhaps, of the results obtained from the various vestibular tests. For example—" On turning to the right, the patient's nystagmus was horizontal to the left of so many seconds, but his past-pointing was so much for the right arm to the right, and so much for the left arm to the right, etc., etc." We all know how hopeless it is to attempt the analysis of a case from such records. To obviate this difficulty, we gradually formulated the accompanying chart, in which all the tests are outlined in the order in which they are usually undertaken, and which is so arranged that when properly filled in, it shows all the vestibular data by a single glance at one page. If this chart is filled in, it will represent a fairly complete examination of the labyrinth, and will contain the findings in practically all the essential tests. We think this chart is of so much help in the examination and diagnosis of a case, that it is actually a part of the technic, and we will therefore describe it.

One side of the chart is devoted to miscellaneous details or such routine matters as are found on any chart, with particular emphasis on examination of the cochlea. The other side is devoted exclusively to the vestibular tests, of which there are three subdivisions—spontaneous, turning and caloric. There is a complete column for the "nystagmus" and another complete column for "pointing"; this enables us to study the nystagmus *as such* by running the eye down the column to the left, showing first the spontaneous nystagmus, then the nystagmus after turning, and then after the caloric test. In the same way the pointing tests can be studied *as such* by following right down the page, first the spontaneous, then those after turning and then those after douching.

As a further aid there is a space reserved on the chart for summarizing the findings obtained after each form of examination. As we always look for the following phenomena—nystagmus, vertigo, past-pointing and falling—they are indicated concisely one underneath the other in each of the three subdivisions. The examiner, after he has turned the patient, simply summarizes by indicating "nystagmus normal" or "absent"; vertigo "normal" or "subnormal," or "exaggerated" or "absent" as the case may be, and so on in the same way under the other headings. When the chart is filled in as we have indicated and an analysis of the case attempted for the purpose of diagnosis, all the examiner need do is to look at these different *summaries* under the three main subdivisions to get a bird's-eye view, as it were, of the whole case.

We will imagine we are examining a patient and conducting the tests step by step with the aid of the chart. The "miscellaneous" side of the chart is filled in first, taking up every heading indicated and filling it in with appropriate information. "DIAGNOSIS" and "SUMMARY" are put at the head of the chart merely for the sake of convenience, so that at any time in referring to a patient's history we have the name and immediately below the principal findings of the examination. The patient's chief complaint is first

CHART I A

**Name** **Age** **Date**
**Address**
**Referred by**

**DIAGNOSIS:**

**SUMMARY:**

**Complains of**

**HISTORY:**
**Dizziness**
**Staggering**
**Deafness**
**Tinnitus**

**NOSE:**

**THROAT:**

**A. D.**
**EARS:**
**A. S.**

**Fistula**

**Hearing Tests**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ \| | A \| | Ac | Bc | n | Pol. \| | $c^4$ \| | Galt \| |
| | | Ac | Bc | n | | | |

noted, as it states at once the purpose of the examination. The history of the case is then taken, paying especial attention to the following:

## CHART 1 B

### TESTS OF THE VESTIBULAR APPARATUS

| NYSTAGMUS | SPONTANEOUS | POINTING | |
|---|---|---|---|
| | | RIGHT | LEFT |
| Looking to RIGHT | Shoulder from above | | |
| Looking to LEFT | | | |
| Looking UP | Nystagmus<br>Vertigo<br>Past-pointing | | |
| Looking DOWN | Falling<br>Romberg<br>Turning head to right<br>Turning head to left<br>Attempt to overthrow | | |
| | TURNING | | |
| To RIGHT<br>Amp.<br>Duration Sec. | To RIGHT<br>Shoulder from above | to | to |
| | Nystagmus<br>Vertigo<br>Past-pointing | | |
| To LEFT<br>Amp.<br>Duration Sec. | To LEFT<br>Shoulder from above | to | to |
| | Nystagmus<br>Vertigo<br>Past-pointing | | |
| | CALORIC | | |
| Douche RIGHT<br>Amp.<br>After min. sec. | Douche RIGHT<br>Shoulder from above | to | to |
| | Nystagmus<br>Vertigo<br>Past-pointing<br>Falling | | |
| Head Back<br>Amp. | | to | to |
| Douche LEFT<br>Amp.<br>After min. sec. | Douche LEFT<br>Shoulder from above | to | to |
| | Nystagmus<br>Vertigo<br>Past-pointing<br>Falling | | |
| Head Back<br>Amp. | | to | to |

From Dr. I. H. Jones' Equilibrium and Vertigo.

Copyright, 1918, by J. B. Lippincott Co.

(1) Dizziness. The patient is asked at what time in his life he first noticed the dizziness. If the dizziness came on gradually or suddenly? Was it a steadily increasing dizziness over several weeks or months or did he have a sudden attack? If he had an attack, did it come on with a sudden change of position, for example when he got out of bed in the morning or when washing the face? Was there nausea and vomiting? Does he have periodic attacks of this sort?

(2) Staggering. Does the staggering come on suddenly or gradually? Is it constant or does it come on intermittently? Was it accompanied by dizziness or did it follow an attack of dizziness? Was the staggering sufficiently severe to cause him to fall at any time? To which side did he fall? Did the staggering take place in any definite direction? In walking did he notice whether he tended to walk toward one side? If staggering or falling are present, is it always in the same direction? Has the feeling of unsteadiness increased or decreased?

(3) Deafness. When was it first noticed? Was it a gradual impairment or a sudden loss of hearing? Did it seem to come as the result of a definite cause, such as a loud explosion, a fall or an illness?

(4) Tinnitus. When was it first noticed? What kind of a noise is it? Is it the sound of bells, the rushing of a railroad train, or is it a sound synchronous with a heart-beat? Are the subjective noises increasing or decreasing?

All other questions as to the history, such as the family history or previous medical history, may be recorded at this point. When dealing with patients of sufficient intelligence, it is a great convenience to ask the patient to write his own history at home; he is instructed to write down in his own way all that he can remember about vertigo, staggering, deafness and noises in the head. Having this done at home not only saves time for the examiner, but elicits a much more complete history, with stress perhaps more correctly placed than in reply to leading questions.

## Nose and Throat

The routine examination of the nose and throat is then made, in order to determine if there is any abnormality which may have a bearing upon the condition of the labyrinth or which could give information of an intracranial disturbance. Such conditions would be suppurative sinus disease, anesthesia of the nose or throat, paralysis of the tongue, paralysis of the vocal cords or absence of taste or smell.

## The Ear

By an examination of the external and middle ear we note the following:

(1) Configuration of the auditory canal.

(2) The presence or absence of a mechanical obstruction in the canal, congenital or acquired; of the latter there may be impacted cerumen or other debris or polypi.

(3) The presence of inflammation or suppuration. If suppuration is present the *fistula* test is made; this can be done with the pneumatic otoscope or the Politzer bag. The ear-piece must be closely fitted into the external canal, preventing escape of air. On pressing the rubber bulb, it is noted whether there occurs any nystagmus or vertigo. These phenomena can usually be elicited only when there is a communication between the external world and the internal ear because of caries of the outer wall of the labyrinth; the fistula test is occasionally positive, however, in cases in which the foot-plate of the stapes is unduly mobile in the oval window.

(4) The condition of the tympanic membrane. The length of time necessary to douche before a reaction appears may depend in a measure upon the thickness of the drumhead. Perforations are looked for, since a "dry perforation" would be a contraindication to douching.

## FUNCTIONAL TESTS OF HEARING

We are now in a position to test the hearing. It is a great privilege to present here for the first time in book-form the simplified method of the functional tests of hearing of B. A. Randall, whose teachings may safely be regarded as the “last word” on this subject. These tests are very simple and briefly recorded on two parallel lines: The upper line represents the right ear—the lower line the left ear. A glance at the chart will show that all the important tests of hearing essential to a diagnosis are made, and yet the tests can be made in a very few minutes. “$\underline{A}$” represents the tuning fork of 50 double vibrations per second. This tuning fork is set in vibration and brought toward the ear, end on; the distance from the ear at which the patient first perceives the sound is noted alongside of this letter. The “A” not underlined represents the tuning fork of 200 double vibrations. To impart to this fork a stroke of standard strength, it is placed vertically upon the middle of the thigh and the tines are allowed to drop of their own weight against the muscular cushion of the knee; it is then brought, end on, toward the ear, and the distance at which it is first heard is noted. C4 represents a tuning fork of 2000 double vibrations. This fork is not used for distance, but it is noted whether the hearing of this fork is good, fair, poor or none. Ac represents “air conduction.” Bc represents “bone conduction.” N represents “normal.” For this test the A fork is used (of 200 vibrations) and it is noted whether this fork is heard better held in front of the ear, or when the stem is held against the mastoid process; the patient is merely asked whether it is louder “front” or “back.” If the sound is heard better in front of the ear than when pressed against the bone, this is represented by the algebraic sign “greater than” ( > ), and is recorded as Ac > Bc. The tuning-fork is then pressed against the mastoid process and the patient is asked to say “gone” at the moment when he ceases to hear it; he should hear it as long as the examiner can feel the vibrations of the fork with his fingers. If the patient says “gone” at the moment that the examiner ceases to feel it with his fingers, the result is recorded as Bc equal to N ($\text{Bc} = \text{N}$). If he says "gone" before the vibrations have ceased, it is recorded by the algebraic sign, less than, namely $\text{Bc} < \text{N}$. If, on the other hand, the patient continues to hear it after the examiner has ceased to feel it, it is recorded Bc greater than N ($\text{Bc} > \text{N}$). Galt. represents the Galton whistle. By starting at the topmost portion of the whistle, the barrel is slowly turned, cautioning the patient to tell the examiner when he hears not only the puffing sound, but also the distinct *squeak*. The number is read on the barrel and recorded. Pol. represents the Politzer test. The 200 fork is set in vibration and held in front of the nostrils. The patient is asked to swallow. If on swallowing he hears the sound louder, it indicates a normal opening of the eustachean tubes in the act of swallowing. This is recorded as Pol.+. The Weber test is the last used; the tuning fork is set in vibration and placed on the vertex of the head or in some other position midway between the two ears, and the patient is asked in which ear he hears it louder. If he hears it louder in the right ear, a capital "W" is placed on the upper line; if he hears it louder in the left ear, a "W" is placed on the lower line.

In these two lines then we have the information to determine the following:

(1) The approximately lowermost range of hearing, the median range of hearing, and the highest range of hearing.

(2) The relation of air-conduction to bone-conduction.

(3) The duration of bone conduction.

(4) The patulency of the eustachean tubes.

(5) The lateralization of sound.

In other words, we have determined all the essential features necessary to make a diagnosis of the auditory apparatus as well as determining, if any deafness exists, whether it is an obstructive lesion of the conducting apparatus of the ear or a percipient lesion of the internal ear. And yet the entire series of tests consumes only a few minutes, and the only instruments used are three tuning forks and a Galton whistle.

## EXAMINATION OF THE VESTIBULAR APPARATUS.

We are now ready for the vestibular tests, all of which are outlined on the "vestibular" side of the chart.

### SPONTANEOUS PHENOMENA

#### NYSTAGMUS

The patient is instructed to look straight ahead of him at a distant point. It is then noted if he has any spontaneous nystagmus. The effort at convergence when looking at a near object will obscure the findings, as it is capable of limiting or entirely effacing a nystagmus. Opaque spectacles may be used, and the eyes observed behind them by looking at the eyes from above. Bartels first suggested the use in this connection of convex lenses. These have a double advantage; in the first place, the observer is able to view the eyes from in front; in the second place, the eyes are greatly magnified and any slight nystagmus is more easily observed. These convex lenses are so powerful that they prevent accommodation and convergence and thus accomplish the same results as looking at a distance.

The patient is now told to look to the right, to look to the left, up and down. This may be accomplished without help from the observer, provided the patient is intelligent. It is also helpful, however, for the examiner to hold his finger at a distance of two feet away from the eyes of the patient and to move the finger to the right, to the left, up and down, and have the patient follow the direction of his finger. Another simple method for examining for spontaneous nystagmus is to place the patient in the revolving chair, have him fix his eyes on some distant point and then turn the chair first to the right and then to the left. In this way we revolve the body, as it were, around the eyes; this is very useful in stupid individuals or in patients who are weak or irritable. All the patient is required to do is to keep looking at some particular object. By any of these methods it is observed whether any nystagmus occurs when the eyes are looking to the extreme right or the extreme left. The patient is then told to look up at the ceiling and down at the floor and it is noted if any nystagmus occurs in these extreme positions of the eyes. At the same time, also, when the patient is looking in different directions, we observe any paralysis or paresis of the eye muscles. If there be any limitation of movement of the eye or eyes, it is of course best when possible to have the patient examined by an ophthalmologist; but it is necessary for the otologist to familiarize himself with the spontaneous action of the eyes in any given case before he undertakes the vestibular tests.

In examining the eye for nystagmus, it is essential to have a good illumination. Occasionally, it is helpful to use reflected light from a head mirror. In order to have a good view of the eye-ball, the upper lid may be raised by the thumb of the examiner. In examining for a horizontal nystagmus, the patient is directed to look straight ahead; the examiner can note the most minute twitches by observing the inner margin of the cornea, at which point there is a glistening light-spot where the cornea curves into the surface of the sclera. In examining for a rotary nystagmus, it is often helpful to have the patient look occasionally at the floor; this reveals the upper part of the sclera and the little vessels radiating in the sclera will then show the slightest rotary movement. Under all circumstances the examiner must remember to close the eye occasionally by pushing the upper lid down with his thumb and then lifting it up again; otherwise the drying of the conjunctiva which occurs very quickly is unpleasant to the patient.

Nystagmus is recorded by means of an arrow—a straight one for horizontal nystagmus and a curved one for rotary nystagmus. It seems simpler, as suggested by George Mackenzie, to have the arrow point in the direction in which the examiner sees the nystagmus *on the patient.*

## Spontaneous Vertigo

The patient is asked not merely whether he is "dizzy," but whether he has any sensation of turning. If the latter is present he is asked to note carefully whether the turning is *systematized* or not. By systematized vertigo is meant, (1) Does he feel he is going in a definite direction? (2) Does he feel that *he* is not moving but the outside world is going in one direction around him? (3) Does he feel that *he* is going in one direction and the outside world is going in another direction?

### SPONTANEOUS POINTING

The object of the test is explained to the patient, namely, that with closed eyes he is to feel the finger, raise his arm to the vertical position and come back and try his best to find the finger again. It is important not to have him move the arm too fast. From the instant that he leaves the finger until he comes back to it again the movement should occupy about two seconds. The results are recorded under the pointing column. Pointing of the right arm is recorded under the word "RIGHT" and the pointing of the left arm under the word "LEFT." If the patient's arm deviates either to the right or to the left, the test is repeated a number of times to make sure that the failure is not the result merely of pure inattention, but that the deviation is constant and persistent in a certain direction. The distance of the deviation is recorded in inches either to the right or to the left as the case may be, by noting under the appropriate column—let us say "2 to R.," if the deviation happens to be two inches to the right, or "2 to L.," if the deviation happens to be to the left, etc. If the patient fails to find the finger and past-points either to the right or to the left, his arm must never be pulled over towards the examiner's finger, if the test is to be repeated, because if this is done the patient finds out that he has "past pointed" and might then make a conscious correction to overcome his actual tendency to past-point. Instead the examiner should again place his own finger under that of the patient. In taking the pointing, it is usually sufficient to test the pointing only of the "shoulder from above," as described; the other forms of pointing, such as "shoulder from below" or "shoulder from the side" are not performed routinely—these are undertaken only in cases where such extensive examinations appear to be necessary.

### SPONTANEOUS FALLING

The Romberg test. The patient is told to stand with the heels and toes close together and close his eyes. Any swaying or falling is noted, particularly the direction of the falling, if any is present. The Romberg test is merely a refined method of testing for spontaneous falling.

Turning head to right and turning head to left. This is done at the same time after taking the Romberg test; the patient is asked to turn his head sharply to the right and then to the left and the examiner observes whether the direction of falling is changed or whether the amount of swaying is increased, with the head in these different positions.

The "attempt to overthrow" is Barany's pelvic girdle test.

Figures 85, 86, 87 and 88 show the pelvic girdle test. The patient stands as before with the heels close together; the examiner grasps the shoulders of the patient and attempts to overthrow him either to one side or the other, forward or backward. The patient is told to balance himself so that he will not fall. When the shoulders of the patient are pressed toward the right, the pelvis should sway toward the left in the attempt to maintain equilibrium. In this way the flexibility and degree of freedom of the pelvic movement is observed.

The goniometer is a platform on which the patient is placed. This platform is so arranged that it can be tilted gradually out of the horizontal plane. It is a refined method of testing the tendency to fall in a given direction. The pictures show the method of using the goniometer. The goniometer shown in the pictures was made by S. A. Brumm. Fig. 89 shows the patient facing the examiner, and standing on a board supported in the centre by braces and at one end by an upright board. The platform on which he stands is supported at these two points. At the other end a rope is attached which runs through a pulley immediately beneath. When the examiner pulls on this rope, as is shown in the picture, the platform is tilted. At the other end of the platform there is a strong spring, which pulls the platform

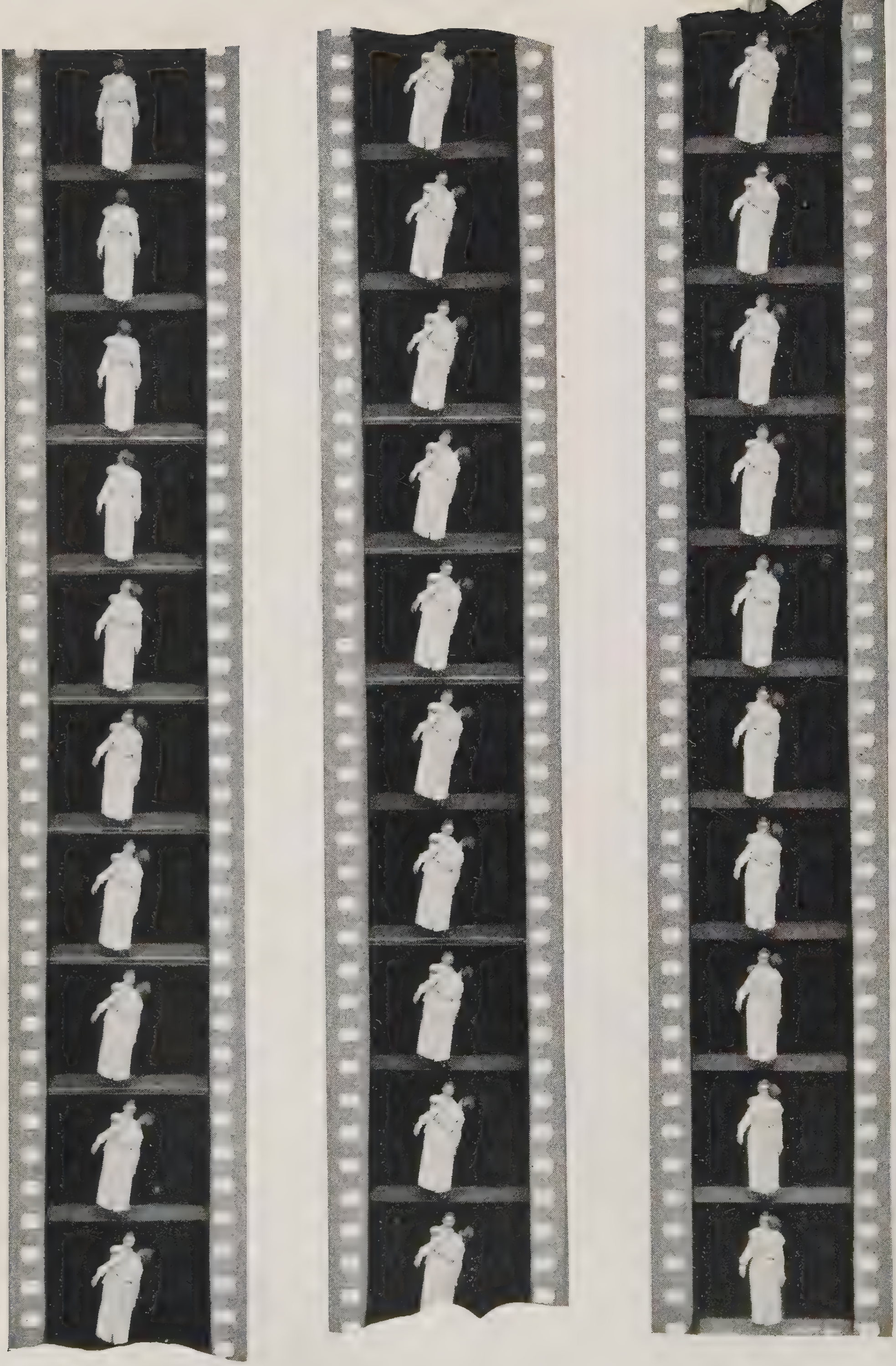

FIG. 85.—Barany's pelvic girdle test.

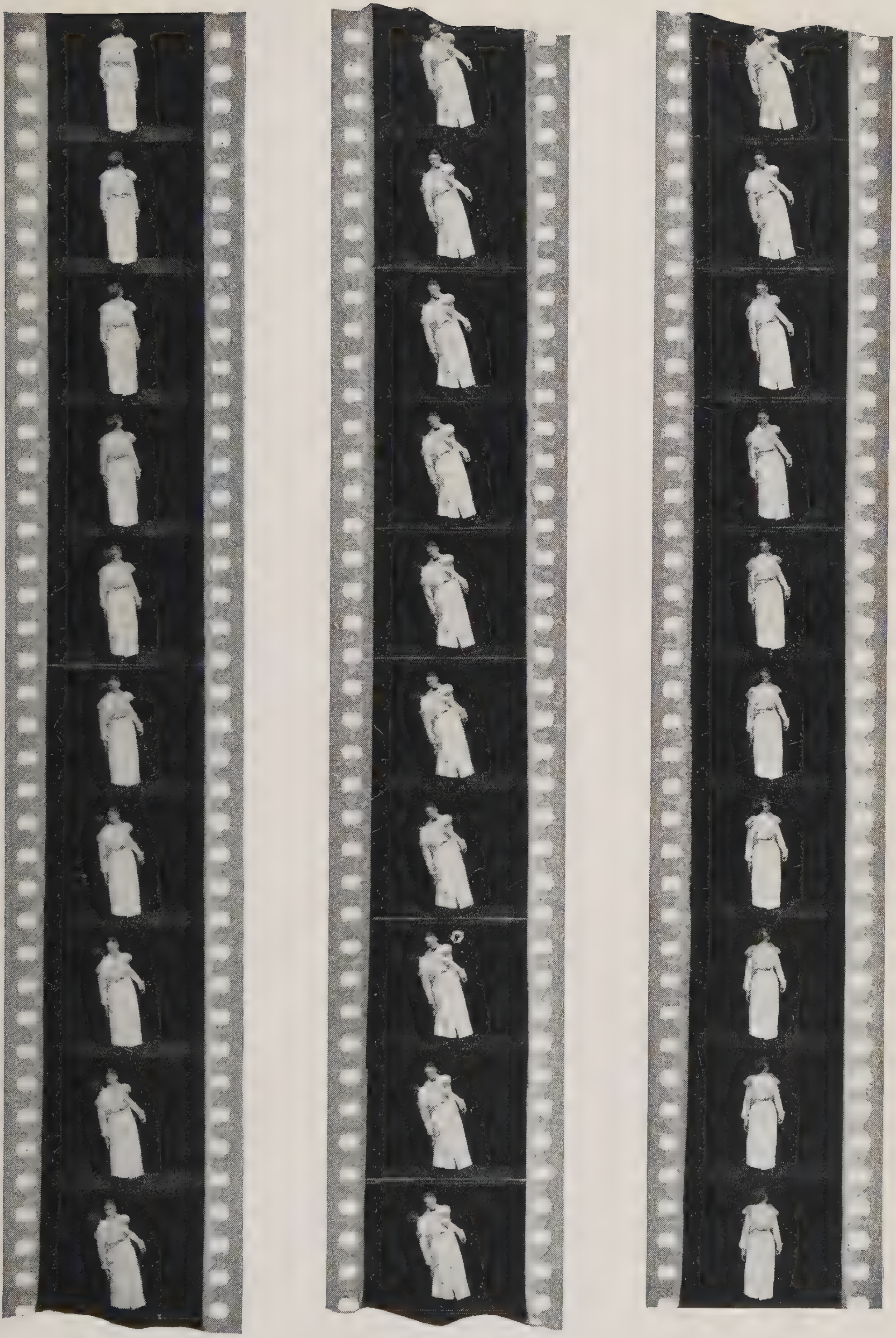

FIG. 86.—Barany's pelvic girdle test.

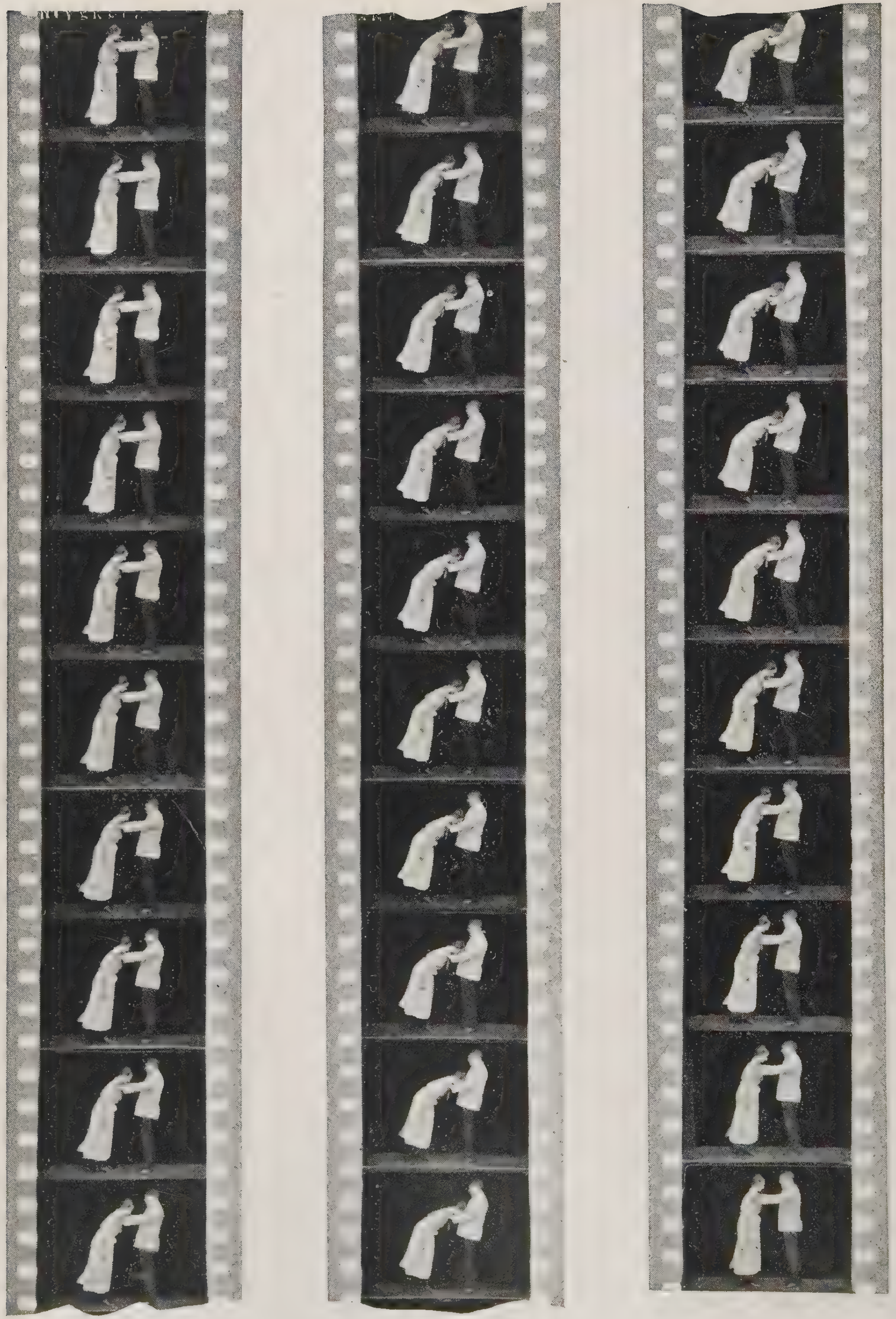

FIG. 87.—Barany's pelvic girdle test.

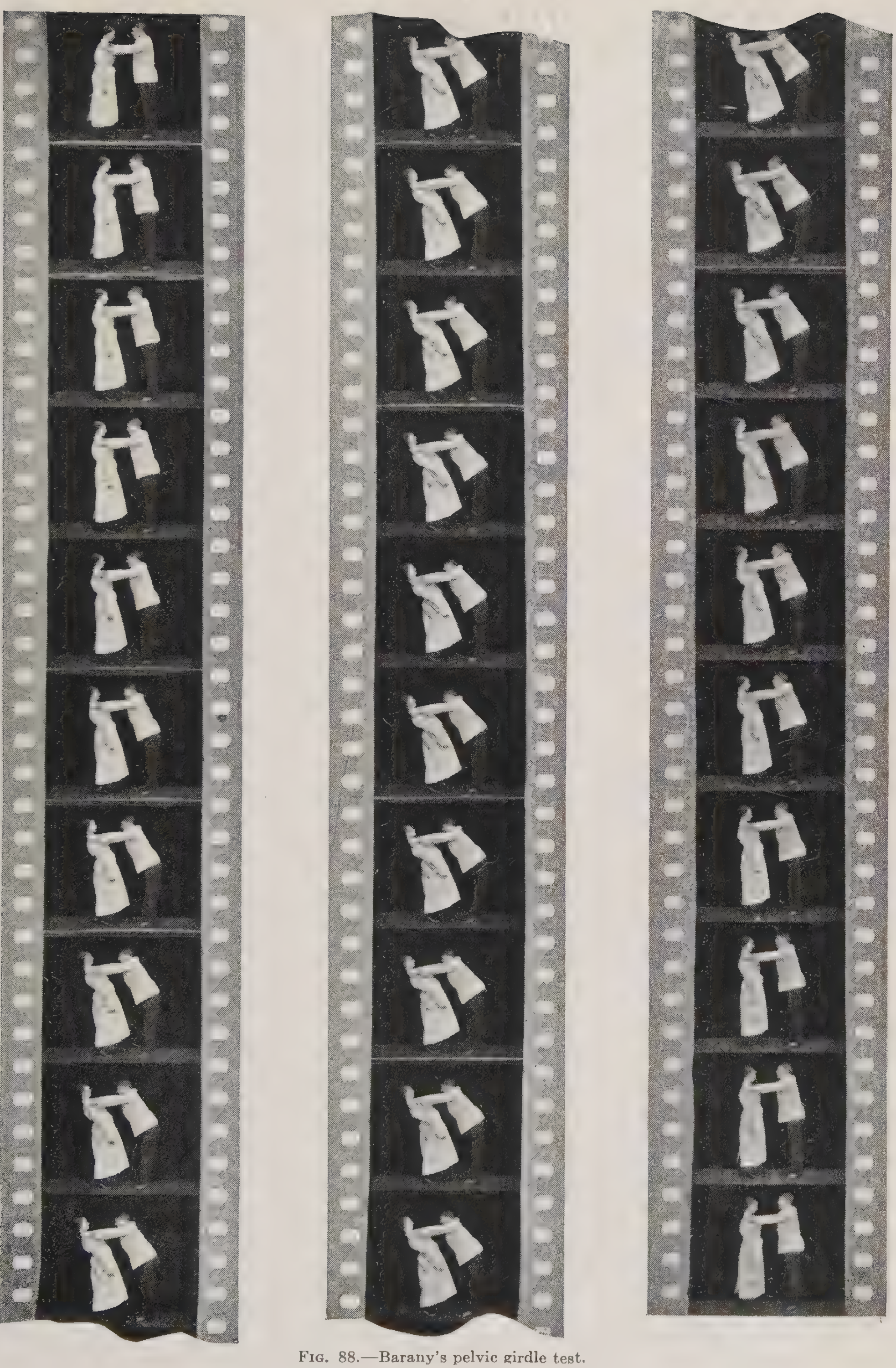

FIG. 88.—Barany's pelvic girdle test.

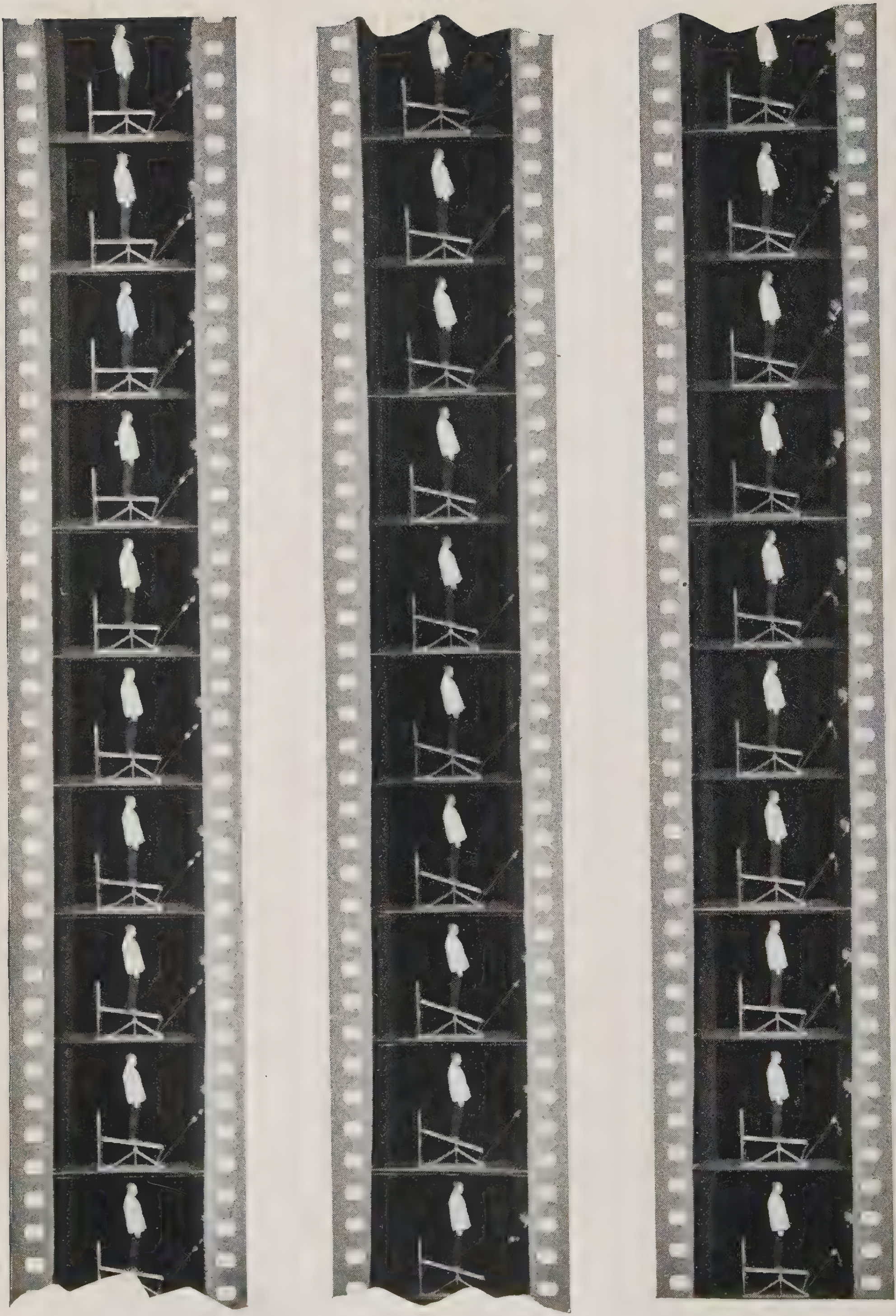

Fig. 89.—Goniometer.

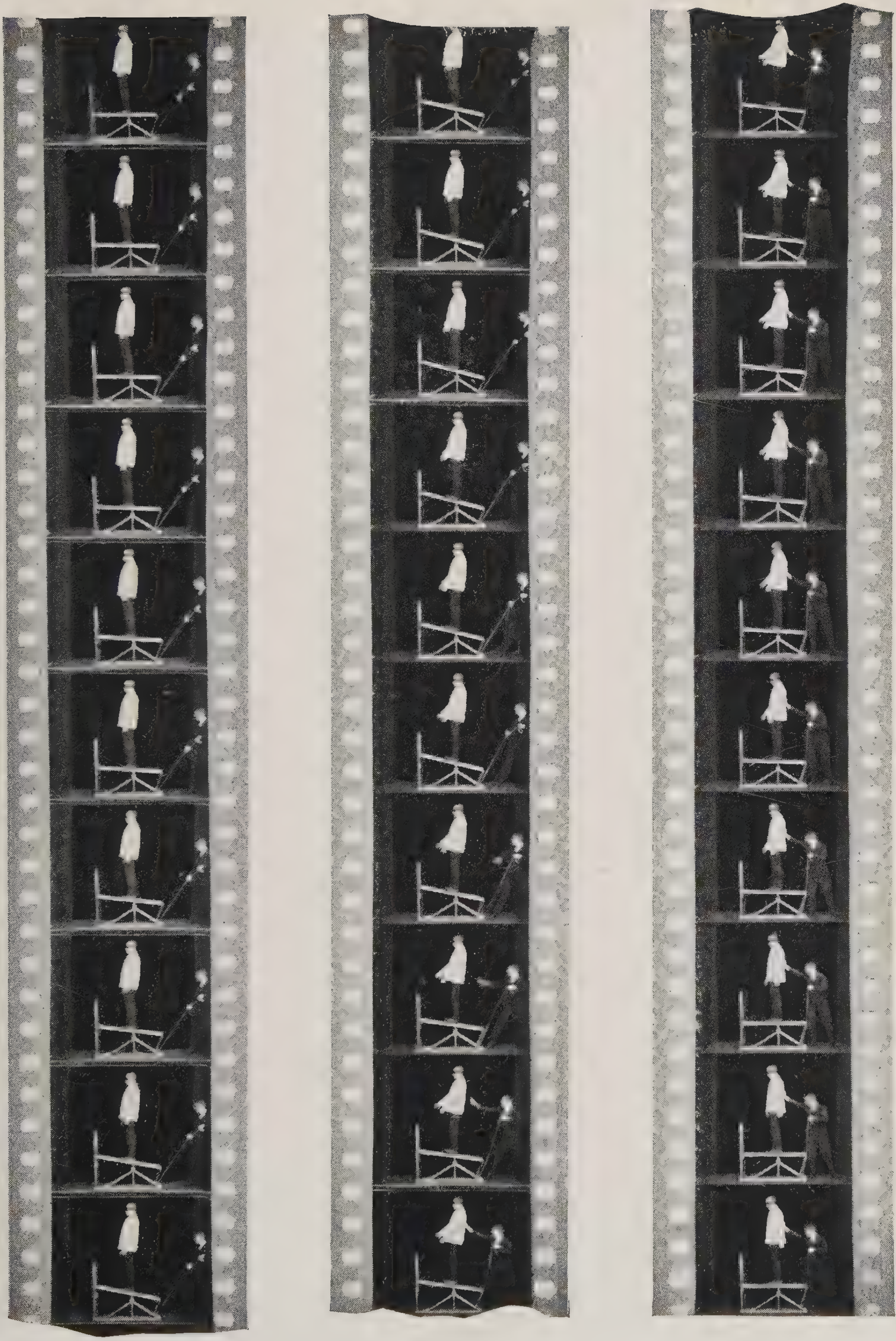

FIG. 90.—Goniometer.

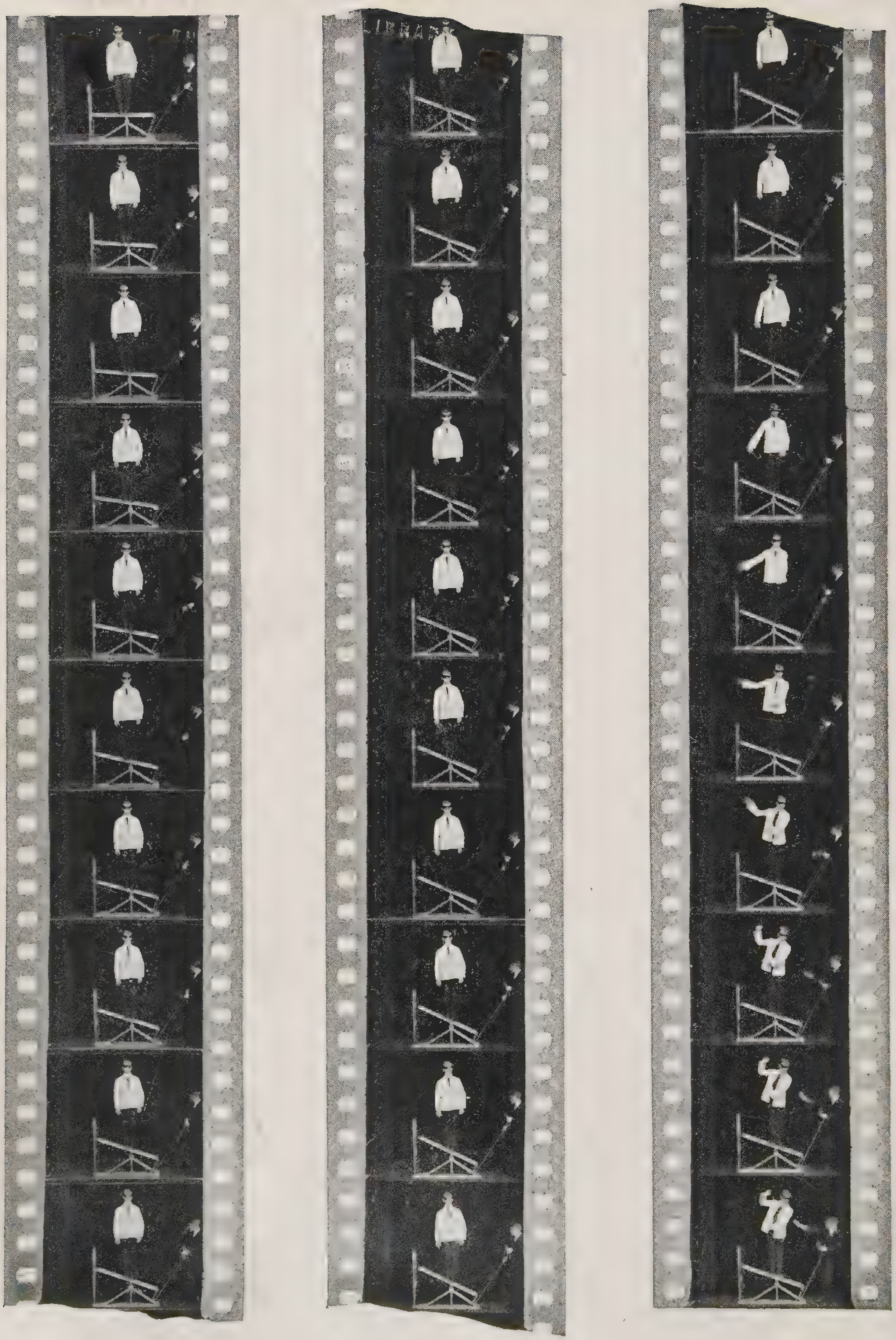

Fig. 91.—Goniometer.

back into the horizontal plane, where it again rests on a support attached to the upright board. This upright board is marked off according to a graduated scale, which shows the exact number of degrees of the inclination of the platform; this can be read off by the examiner at the moment that the patient is unable to maintain his equilibrium. In Fig. 89 the patient is standing in the centre of the platform facing the examiner, who is holding the rope. The patient's eyes are open. The examiner gradually pulls on the rope until he notices that the patient is beginning to sway, whereupon he gradually loosens the rope and allows the platform to go back to the horizontal position. Fig. 90 shows the patient facing away from the examiner. The tests with the patient facing the examiner or turned directly away from the examiner bring out a latent tendency to fall either forward or backward. Fig. 91 shows the patient standing sideways on the platform. These tests would bring out a lateral falling. The more delicate test naturally is with the eyes closed, as the patient is then dependent entirely on the semicircular canals and muscle-sense. By means of the goniometer it is sometimes possible to bring out a latent tendency to fall, not demonstrable in any other way. This, however, is only occasionally useful, and the gonimeter need not be used routinely.

## TURNING

In order to carry out the turning-test with any degree of accuracy, it is essential to have a revolving-chair especially constructed for this purpose. Not only must it revolve smoothly, but it must have the necessary attachments for holding the head in the proper position as well as a stopping device to clamp it instantly and firmly. Barany has constructed a chair for this purpose. He sent us one of these chairs (Fig. 92) which we were fortunate enough to receive only a few days before the outbreak of the war in Europe. So far as we know it is the only one in this country. As it was hardly probable that any more chairs of this kind could come out of Vienna for some time, we were asked to design a chair along

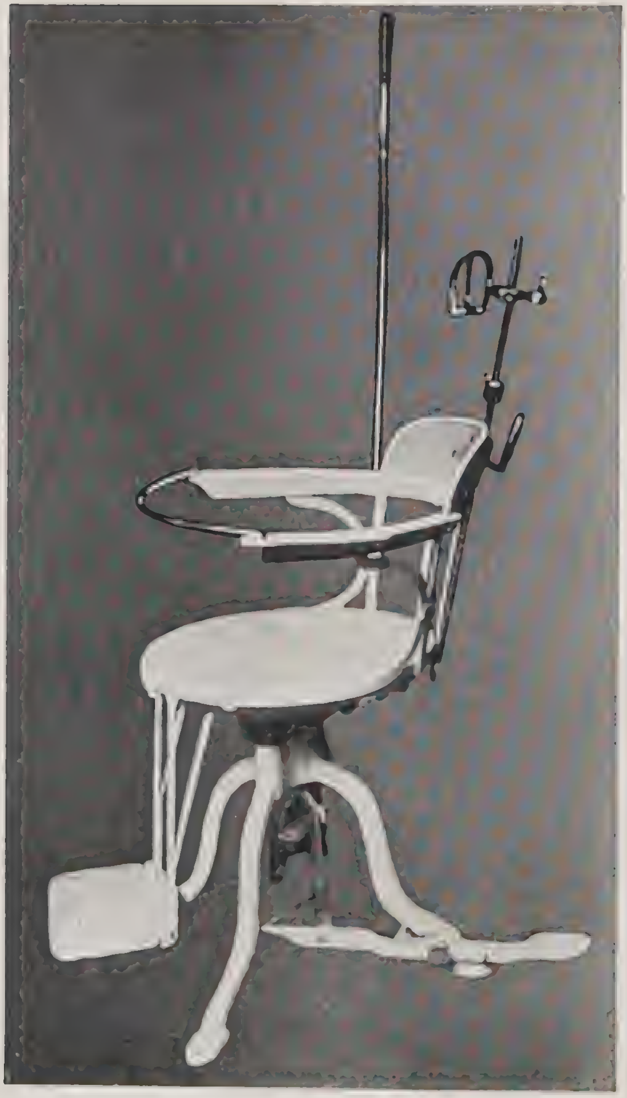

FIG. 92.—Original Barany chair.

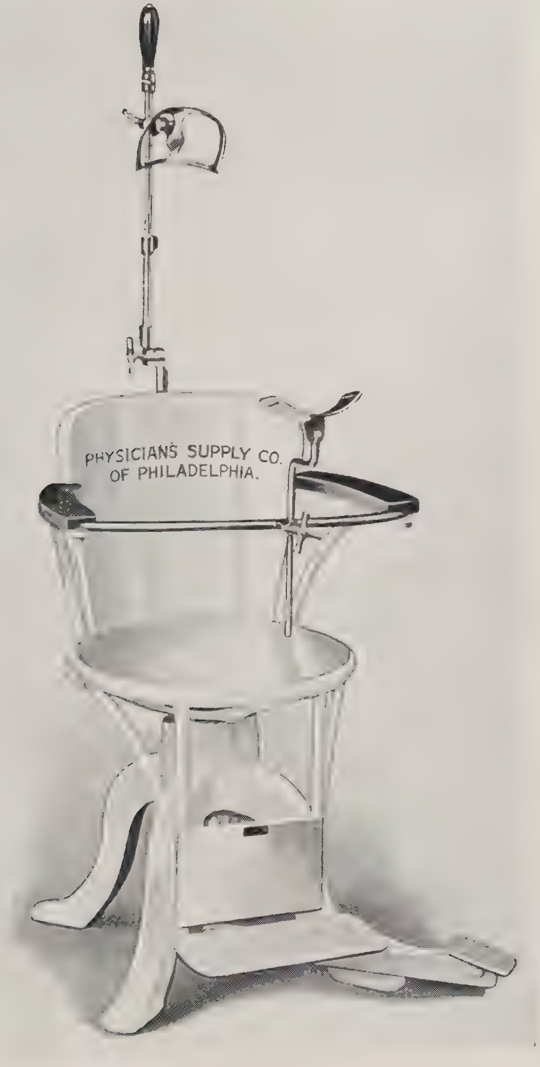

FIG. 93.—Latest modification of American Barany chair.

similar lines. After the Barany chair had been used for a few months, the following changes suggested themselves and were incorporated in the new chair (Fig. 93):

(1) The back of the chair and the head-rest are so constructed that the patient's head is placed immediately over the axis of turning. This is obviously of great importance. In the original Barany chair the head revolves away from the centre of turning and describes a circle with a diameter of over a foot.

(2) It is impossible in the Barany chair to hold the head in a forward position; we therefore constructed an extra headpiece to permit rotation with the head inclined forward. It is obvious that it is absolutely essential for the head to be held steady when being rotated in this position, and this cannot be accomplished without a special head-bracket.

(3) Instead of having a special handle for turning, as in the Barany chair, the rod at the back of the chair is made slightly longer and a handle placed at the top. The extra handle was very annoying and interfered with the pointing tests of the right arm by its presence on the side of the chair.

(4) The base is much heavier than in the Barany chair, in order that the patient may be rotated rapidly, if necessary, and yet not have the chair wobble; this gives both the patient and the examiner a greater sense of security.

(5) The Barany chair is bound together by a great many bolts. In order to take the chair apart, in case it is desired to move it, all these bolts must be undone. In the new chair there are no bolts and the parts are all welded; the chair consists of only two pieces —the seat and the base. This makes it more portable, and is a great convenience when a patient has to be examined at a place where no such chair can be had. In constructing this chair we also aimed to make it suitable for use as a regular office treatment and operating chair, so that it would not require any extra room in the office.

## Nystagmus After Turning

The patient is seated in the chair with the head inclined forward 30°, which puts the horizontal canals in the horizontal plane

(Fig. 94). Eyes closed. The chair is then rotated to the right ten times, at a speed of two seconds to each turn, making twenty seconds for the ten turns, after which it is stopped. The patient keeps his eyes closed during the turning and is told to open his eyes again when the chair is stopped. The after-turning nystagmus is then noted, including its direction, character and duration. It is necessary to time the duration of the nystagmus with a stop-watch. At the instant the chair stops, start the stop-watch. After the last twitching of the eyes has ceased, the stop-watch is again clicked and the time noted. The patient is first turned to the right. After the observation of the nystagmus, it is best to wait a minute, until not only the nystagmus has stopped, but also the subjective sensations have ceased. The patient is turned to the left in a similar manner and the nystagmus noted. Turning in this position with the head 30° forward has tested the horizontal canals.

To test the vertical canals by turning, the head is put forward 120° (Fig. 95). As a matter of fact this forward bend of the head is somewhat difficult at such a marked angle. For all practical purposes the forward position of the head at 90° answers for the stimulation of the vertical canals. After turning the patient ten times and then stopping the chair, we examine the nystagmus either by stooping down low enough to see the eyes of the patient or by lifting the patient's head from the forward head-rest to the back head-rest. Changing the position of the head after turning does not alter the direction of the nystagmus. Another method of examination for the vertical canals is to turn with the head tilted backward 60° (Fig. 96), which produces a nystagmus in exactly the opposite direction to that produced with the head forward. If we desire to produce a vertical nystagmus, the patient's head is inclined toward his shoulder and held in that position by the movable head-rest. After stopping the chair the nystagmus can be examined with the head maintained in this position, or brought upright. The type of nystagmus is recorded in the nystagmus column, the number of seconds noted, and the amplitude recorded in terms of either "Large," "Small," "Fair," or "Barely a twitch."

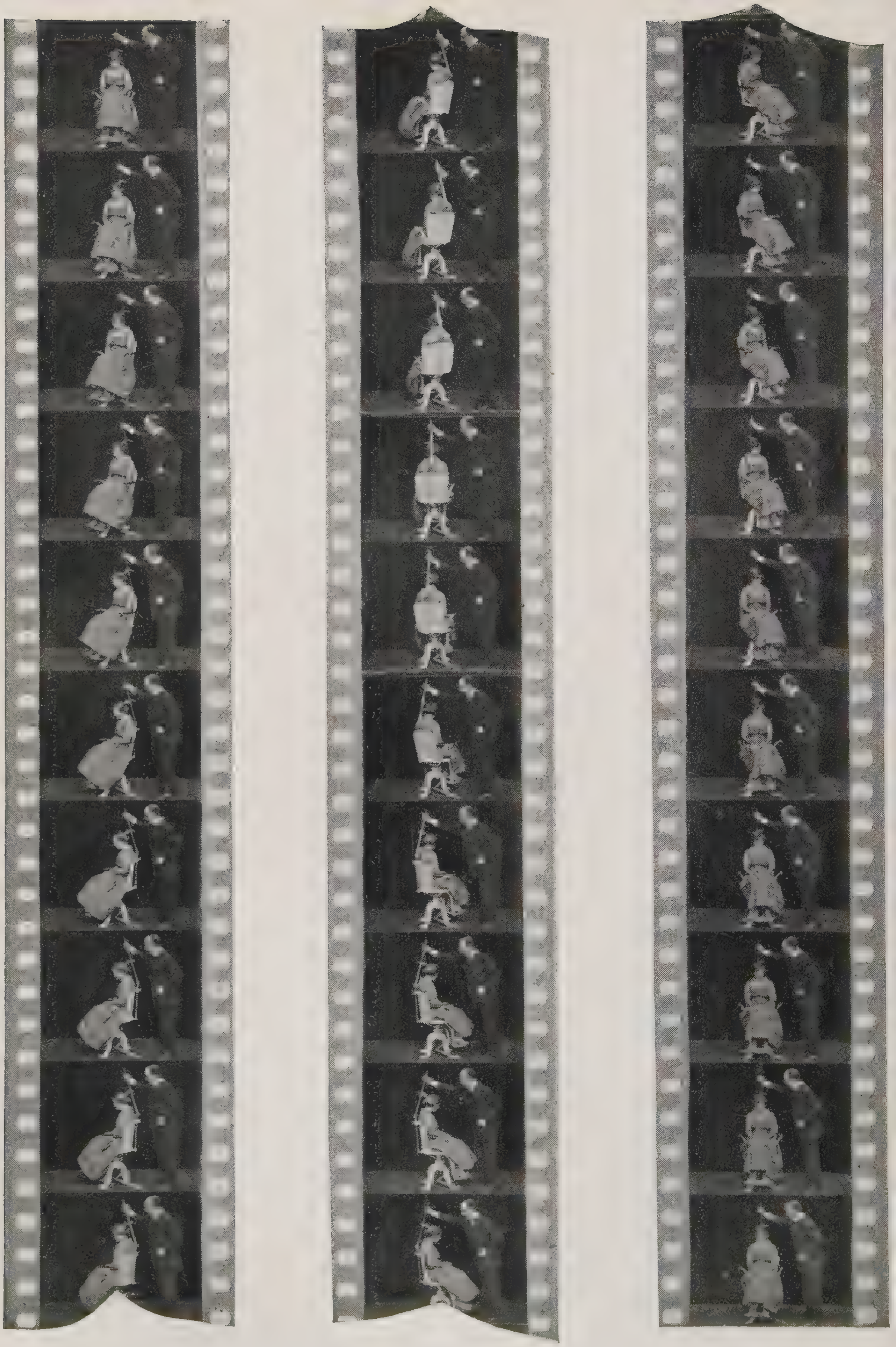

FIG. 94.—Turning to the right, head 30° forward, stimulating both horizontal canals.

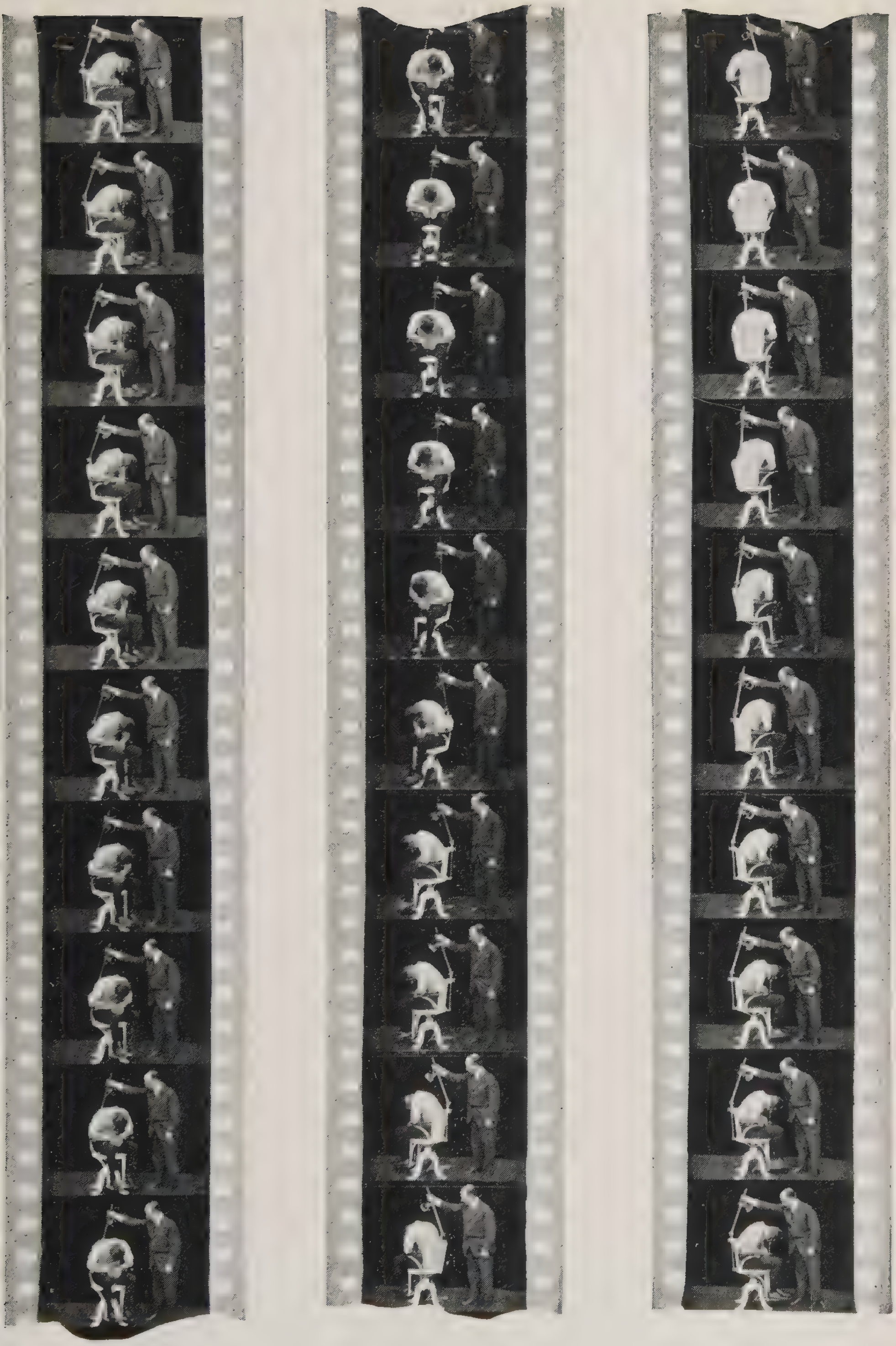

FIG. 95.—Turning to right, head 120° forward, stimulating vertical canals of both ears.

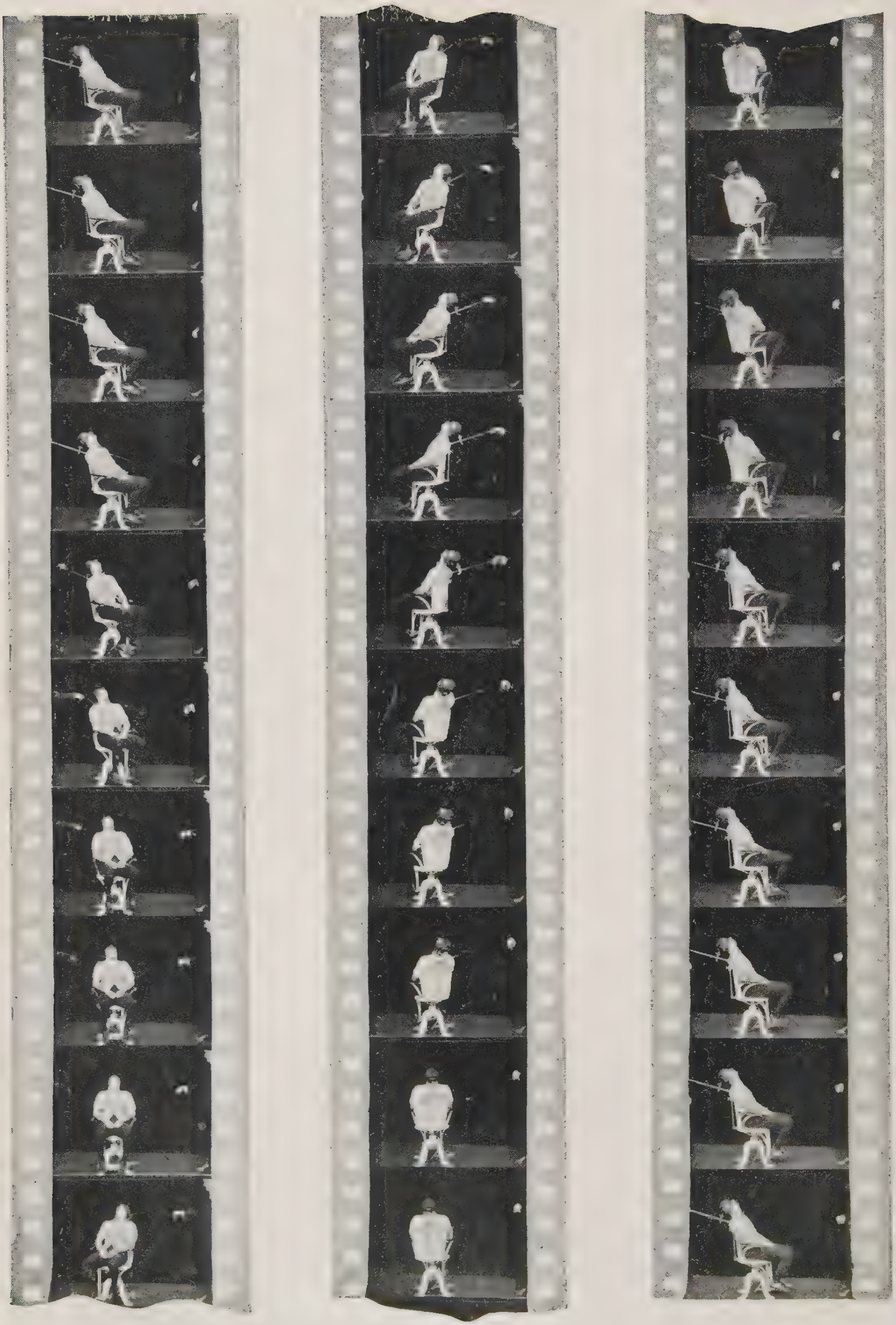

Fig. 96—Turning to the right, head 60° back, stimulating the vertical canals of both ears.

### Vertigo After Turning

As a rule it is not necessary to turn the patient all over again merely for this test. The past-pointing, when it is tested routinely, of itself gives us the evidence of the subjective vertigo. If a more accurate determination is desirable, however, the vertigo after turning may be tested quantitatively. The patient, with eyes closed, is turned to the right at a speed of one second to each turn and is asked to keep on telling the examiner in which direction he is being turned. Thus he keeps on saying "To the right—to the right." After ten turns in ten seconds the chair is stopped and immediately he will say "I am going to the left—to the left." The stop-watch is started at this instant and kept running as long as the patient *thinks* he is going to the left. When he says "I am standing still," the watch is stopped and the reading of the duration of the vertigo taken in seconds. This is the method for the quantitative estimation of vertigo after turning.

### Past-pointing After Turning

The patient is turned to the right at a speed of one turn to the second. That is to say, ten turns in ten seconds. During the tenth revolution of the chair, the foot-pedal is released simply by touching it with the foot. The chair when it reaches the original front position is then clamped rigidly. Naturally the patient must not receive a jolt as the chair is clamped. Therefore during the last half of the final turn the examiner first releases the foot-pedal and then grasps the chair and gradually stops it, allowing the clamp to fasten on it gently. The right index finger is then seized by the examiner's left hand and placed on the examiner's right forefinger. The patient's finger is placed firmly against the finger of the doctor. Once the fingers are approximated, the doctor says "up," the patient then raises his arm to the perpendicular and immediately comes down again, trying to find the finger. The left forefinger is then immediately seized by the examiner and the left arm tested in a similar way. The right arm is then again tested. The left arm is tested. This testing of one arm after the other is continued until all past-pointing ceases. In this way we test the duration of the past-pointing stimulus. In other words we not only determine the extent of the past-pointing of each arm, but also how long the past-pointing continues before the patient is again able to find the finger of the doctor. The extent of the past-pointing of each arm is recorded in inches on the chart.

It is sometimes helpful when seizing the patient's finger to rub one's finger back and forth quickly; this impresses the patient with the exact position of the finger and can easily be done in a second of time.

In cases in which there is a short-lived stimulus, as in lesions of the labyrinth or VIII Nerve, there is frequently an immediate wide past-pointing of the right arm, but the left arm, tested immediately afterwards, touches and shows no past-pointing. In such a case, before suspecting that there is an absence of past-pointing of the left arm, it is necessary to turn all over again and then test the left arm first. If the left arm past-points properly, then the right arm is tested again, when it will be found that the right arm fails to past-point. This naturally shows that the failure of past-pointing is because the stimulus died out quickly and not that there was a disturbance of the past-pointing itself.

As a rule it is sufficient to test only the "shoulder from above." If, however, we wish to test the other joints, the technic is the same. For instance, after turning and clamping the chair, the foot is seized and the big toe is brought up in contact with the finger of the examiner, the patient then lowers the foot and immediately brings it up again in the attempt to find the finger.

In testing for the past-pointing of the arms, it is advisable not to bring the patient's arm back to the median line after he has once past-pointed. It is better for the examiner to place his finger beneath the past-pointing finger of the patient, and again testing, see if there is still further past-pointing. The obvious reason for this is that pulling the arm back again to the median line informs the patient that he has past-pointed and may modify his further attempts to find the finger.

It is usually sufficient to record the amount of past-pointing by the examiner's judgment of the distance in inches as he sees it. It is sometimes however of importance to note the exact degree of past-pointing. For this purpose we use the Victor Horseley screen, modified by Mills. The screen is divided off into one-half inch squares. It is placed in a horizontal position immediately beneath the finger of the examiner. The patient touches the doctor's finger as usual, raises it and brings it down right on the screen. The number of squares is then counted between the finger of the examiner and the finger of the patient. This gives an exact measure in inches of the amount of past-pointing.

On stopping the chair after turning, it frequently happens that the patient will lean to one side or at least change the position of his head. It is advisable in this instance for an assistant to take hold of the head and hold it in the proper position in the head-bracket before taking the pointing-tests. Of course this is all done in a moment's time, so that the pointing-tests may be taken practically at once after the cessation of turning.

It must be remembered, of course, that the eyes are kept shut throughout this entire test. It is sometimes difficult for a patient to remember not to open the eyes. Of course he may be blindfolded, but it is generally sufficient to tell him "do not open your eyes the rest of the afternoon until you are told to." It is usually possible to obtain a sufficient past-pointing of both arms on one turning, but if a comparison between the two arms is advisable it is best to make a complete ten turns for the right arm alone, then to turn him all over again for the pointing of the left arm.

## Falling After Turning

In order to produce falling it is of course first necessary to produce a subjective sensation of movement in one of the *vertical* planes. This is produced by stimulation of the vertical canals, either (1) with the head forward or backward, which produces vertigo in a frontal plane, or (2) with head inclined 90° toward the shoulder, which produces vertigo in a sagittal plane. The usual method is with the head forward. The patient is turned to the right and after ten turns the chair is stopped. The head is then raised to the vertical position and it is noted in which direction the patient falls.

If no falling is produced we can make further tests: Supposing that there is a lesion of the VIII Nerve or labyrinth so that the turning produces very little effect. Under these circumstances, we want to bring out to the fullest extent any latent tendency to fall. This may be done by turning the patient with the head forward and then on stopping, the patient is told to get right out of the chair and do the Romberg test. This will bring out even the slightest tendency to fall.

## Caloric Test

The main advantage of the caloric test of Barany is that it enables us to examine each internal ear separately and also to analyze the function of its canals separately, whereas turning stimulates both labyrinths at the same time.

Temperature of water. It is essential to have an absolute standard. As Barany directs, we employ water at 68° F. in every case—not a degree below nor a degree above. This temperature is sufficiently cool to secure a good reaction, and yet is not so cold as to be uncomfortable to the patient. It is rarely advisable to use hot water; it is much more uncomfortable for the patient and does not give so good a reaction as the cold. We use temperature 112° F.

The vessel containing the water is placed at a position two feet, approximately, above the level of the ear to be douched. The height of the vessel is of no importance whatever—the only matter of real importance is that the stream shall not be so powerful against the drum membrane as to be uncomfortable or injurious. It is only essential that a continuous stream of water shall flow against the drum membrane maintaining an *even temperature*. The shape or size of the nozzle is immaterial. Any nozzle that will extend a short distance into the auditory canal is satisfactory.

For little children, for example, we attach the nozzle of the ordinary ear syringe used for removing impacted cerumen. It is convenient to have a cut-off arrangement in the nozzle itself, although this can be regulated nicely merely by squeezing the rubber tubing between the fingers.

The amount of water is of no importance whatever, as it is not the force or volume of water that produces the reactions, but merely the maintaining of a uniform *coldness* in the ear.

The patient's head is placed in the upright position (with the head tilted forward 30°), just as for turning. This puts the vertical canals in the vertical position. The chair is held firmly by the foot clamp. Stop-watch ready. The nozzle is inserted into the canal and at the instant at which douching is started, the stop-watch is clicked. It is possible for the examiner to conduct the examination entirely alone. On starting the douching, he clicks the stop-watch and puts it in his pocket, holding the nozzle in the ear with one hand, and elevating the eye-lid with the other hand. In this way he watches for the nystagmus to appear. When satisfied that a rhythmic nystagmus has started, he clicks the stop-watch, which he still leaves in his pocket to be examined later, and then, laying aside the douching nozzle, he tells the patient to close his eyes, and then undertakes the pointing tests. It is far better, however, to have a trained assistant in order to do the most careful work; for example, it is best for the examiner to confine all his attention to the douching, in order to make sure that there is a continuous flow of water against the drum-membrane. The assistant takes care of the stop-watch and watches the eyes for nystagmus.

On the instant that it can be said that there is a definite rhythmic nystagmus, no matter how slight in amplitude, the stop-watch is clicked and the exact time noted. It is our custom to continue the douching for a few seconds longer in order to obtain a good reaction. In perhaps five or ten seconds in the normal, from the onset of the nystagmus, the amplitude becomes large. The direction of the nystagmus, its amplitude and rapidity are then noted.

The patient is then told to close his eyes and the pointing tests are carried out. First the right arm, shoulder from above, then the left arm, shoulder from above, are tested. If advisable, the other extremities or the neck and trunk may be tested. The duration of the average stimulus after douching is long enough to permit of the examination of several parts without repeating the douching. In this connection it must be remembered that by douching with the head upright we have produced a rotary nystagmus in the frontal plane, and that there is also a marked tendency for the patient to fall. It is therefore often advisable for an assistant to hold the head of the patient firmly in the head-rest before taking the pointing tests.

Douching with the head backward 60°. Bruenning recommends that the head not only be placed backward, but then tilted towards the shoulder of the side douched. This is not essential but produces a quicker reaction, because there is the anatomical position for the greatest drop of the endolymph. As a rule, it is not necessary to perform the douching test all over again simply to determine the function of the horizontal canal. The ear is douched with the head 30° forward, and as soon as the nystagmus and past-pointing have been noted, the head is quickly tilted to a position 60° back, which places the horizontal canal in the position in which it is affected by the chilling. The patient is told to look upward and the existing rotary nystagmus immediately becomes horizontal. The pointing of both arms is then quickly taken with the head in that position. The head may then be tilted forward 120° and the past-pointing of both arms be taken again. This new position of the head again influences the horizontal canal, but has reversed the direction of the endolymph movement. All the responses, therefore, are also reversed.

The caloric test, by its very nature, produces vertigo only in a plane at right angles to the floor, so that "falling" always occurs on douching a normal ear. All that is routinely necessary is to observe the tendency to fall, while the other examinations are going on, or, after completing the tests with the head in different positions, to have the patient stand and do the Romberg test.

Of course, before douching the ear, it is absolutely necessary to make a careful examination of the drum-membrane and canal. In the case of a healed otitis media with a permanent perforation, it is obviously unwise to douche at all, as there is a grave likelihood of lighting up the old trouble. In such a case we can either rely entirely upon the turning or we can use the galvanic test; if, however, the caloric test would be of unusual importance, there may be used an apparatus which would introduce cold air against the drum-head. Various types of apparatus for this purpose have been devised by Ruttin, Bloch, Dundas Grant and Aspisoff. Cold air is, however, rather unsatisfactory. Then, again, if we are making the pointing tests and we wish to bring out the pointing of both arms in both directions, we can easily accomplish this by douching the opposite ear, first with cold and then with hot water. In this way we leave the ear that has the dry perforation as much alone as possible.

## Electrical Test

For practical purposes the electrical test has, unfortunately, a very limited usefulness. Since the ability to examine one set of canals at a time is of such prime importance in making these tests of clinical usefulness it is evident how limited are the uses of an agency which stimulates at the same time not only the entire labyrinth but the VIII Nerve as well and perhaps even the medullary nuclei themselves. Also whereas the normal person responds to a current between 4 and 6 milliamperes (m.m.), which is not painful, pathological cases require such a strong current that it is distinctly painful. Fortunately the caloric test, by fulfilling all requirements, makes the electrical test unnecessary in the average case. The one use to which the electrical test may be applied in which it is very useful is in making a differential diagnosis between involvement of the labyrinth and VIII Nerve. In attacking this problem the electrical test is the only means of differentiation. In a recent destruction of the labyrinth, the caloric test will fail to produce responses; the electrical current, however, will directly affect the intact VIII Nerve and produce normal reactions.

The galvanic current is used. A large electrode is held in one hand, a small one is placed on the mastoid process. Both electrodes should be covered with cloth or cotton and should be kept fairly saturated with a normal salt solution. (This decreases the unpleasantness of the current.) The current is gradually turned on, and when 4 m.m. are discharging a nystagmus should appear. Another method is to have two small electrodes, one applied to each mastoid. In this way the kathode of one side is reinforced by the anode on the opposite ear, in the same way that both ears are stimulated by turning. It is better, however, to apply the current to one ear at a time, just as the caloric is a more accurate test than the turning, in that it examines one ear at a time. The quantitative measurement of nystagmus is made by noting what strength of current is required to produce it. If the anode is placed over the right ear, it gives the same reaction as cold water douched in the right ear. The kathode gives the same response as hot water douched in the right ear. That is to say, the anode on the right ear produces a rotary nystagmus to the left, the kathode on the right ear produces a rotary nystagmus to the right. Unlike the caloric test, however, the reaction lasts only so long as the current is applied.

It must not be thought that a routine examination of the internal ear requires all the preceding tests. In the average case it is necessary merely to fill out one chart and the examiner then has before him all the essential data of a routine examination.

# CHAPTER XIX

## EXAMINATION OF A CASE WITH THE USE OF CHART

For the convenience of those unfamiliar with these tests, a brief résumé is given of the method of examining a patient and recording the findings on the chart (see Chart II). This chapter may be used as a "ready reference" to be followed until the examiner becomes familiar with the technic.

We will consider the examination of a normal person. On the first page of the chart are recorded the chief complaint and history, the examination of the nose, throat and ears and the hearing tests. The normal responses to the hearing tests are shown on the chart.

On the second page of the chart we now record the vestibular tests. The spontaneous phenomena are first noted. The patient looks to the extreme right and to the extreme left; the normal shows very little or no nystagmus. The patient then looks upwards and downwards, the normal showing no nystagmus. This is recorded on the chart in the nystagmus column, after "Looking to the Right," "Looking to the Left," "Looking Up," and "Looking Down." The patient is then told to close the eyes and the spontaneous pointing tests are taken and recorded under the pointing column. The normal is always able to find the finger and this is recorded by the letter "T," which signifies "touched."

Turning. Place the patient in the chair in a comfortable position and arrange the bracket so that the head is tilted forward 30°. The foot-pedal is released; the patient closes his eyes and the chair is then turned to the right at a speed of two seconds to each turn for ten turns. The chair is stopped, and the stop-watch is immediately clicked. At the same instant the patient is told to open his eyes and look at a distant point. The nystagmus is

## CHART II A

**Name** **Age** **Date**
**Address**
**Referred by**

**DIAGNOSIS:** Normal

**SUMMARY:** Normal reactions, suggest normal -
1. Labyrinths
2. VIII Nerves
3. Medulla oblongata
4. Pons
5. Cerebellar peduncles
6. Cerebellum

**Complains of** Nothing

**HISTORY:**
Dizziness None
Staggering None
Deafness None
Tinnitus None

**NOSE:** Normal

**THROAT:** Normal

**EARS:**
A. D. Normal
A. S. Normal

**Fistula** Negative

**Hearing Tests**

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3 | A | 8 | Ac > Bc = n | Pol. | + | $c^4$ | Good | Galt | .7 | W ± |
| = | 3 | | 8 | Ac > Bc = n | | + | | Good | | .7 | |

# CHART II B

## TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT Very slight or none
Looking to LEFT Very slight or none
Looking UP None
Looking DOWN None

POINTING

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling None
Romberg Negative
Turning head to right Negative
Turning head to left Negative
Attempt to overthrow Normal pelvic movements

---

### TURNING

To RIGHT →
Amp. Good
Duration 24 Sec.

| To RIGHT | | |
|---|---|---|
| Shoulder from above | 12" to Right | 10" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

To LEFT ←
Amp. Good
Duration 24 Sec.

| To LEFT | | |
|---|---|---|
| Shoulder from above | 10 to Left | 12" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

---

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 40 sec.

| Douche RIGHT | | |
|---|---|---|
| Shoulder from above | 8" to Right | 6" to Right. |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | | |
|---|---|---|
| Head Back → <br> Amp. Good | 10" to Right | 8" to Right |

Douche LEFT ↶
Amp. Good
After min. 40 sec

| Douche LEFT | | |
|---|---|---|
| Shoulder from above | 6" to Left | 8" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | | |
|---|---|---|
| Head Back ← <br> Amp. Good | 8" to Left | 10" to Left |

watched, and the instant that the nystagmus ceases the stop-watch is again clicked. The normal person shows a horizontal nystagmus of good amplitude and of 24 seconds duration; the type and direction of nystagmus are recorded by an arrow as shown on the chart after the words "TO RIGHT." Under this are recorded the amplitude and duration. The patient is then turned to the left, just as before, and the nystagmus recorded after the words "TO LEFT," in the nystagmus column.

For the pointing tests after turning, the patient is again turned to the right, but twice as fast as before—one second to each turn for ten turns. When the chair is stopped, the patient keeps his eyes closed and the chair is held firmly by the foot-clamp. The pointing tests are immediately taken, first of the right arm, then of the left arm, then again of the right arm and again of the left arm until he no longer shows any past-pointing. The normal person past-points approximately 12 inches to the right with the right arm and 10 inches to the right with the left arm; this is recorded in the pointing column after "TURNING." "12 to R" means "12 inches to the right." The first pointing of each arm is all that need be *recorded* as a method of routine provided that the past-pointing has proven normal in duration. The normal shows three past-pointings of each arm. The patient is then turned to the left, just as before, and the past-pointing is recorded under the pointing column.

By this turning we have tested the horizontal canals of both ears; the normal nystagmus has demonstrated normal horizontal canals and normal pathways from the horizontal canals of both ears to the eyes. The normal past-pointing has demonstrated, *per se,* a normal vertigo, indicating unobstructed pathways from the horizontal canals of both ears through the cerebellum to the cerebrum; also normal pathways from the cerebral motor cortex through the brain-stem and cerebellum to the upper extremities.

Caloric. The head is *kept in the same position* and the douching then tests the *vertical* canals, but, of course, only of the ear douched. Water, 68°, is introduced into the right ear; at the second the douching is started the stop-watch is clicked. The eyes are watched carefully, and the patient is told to look down at the floor occasionally so that the beginning of the nystagmus may be instantly noted by observing the little blood vessels of the sclera. It is best to elevate the upper lid with the thumb, but it is also wise frequently to press the eyelid over the eye to prevent the dryness of the conjunctiva, which is uncomfortable to the patient. At the instant a definite nystagmus appears, the stop-watch is again clicked. The type and direction of the nystagmus are recorded by an arrow shown on the chart after the words "Douche Right." Under this is recorded the amplitude and by referring to the stop-watch we note how long it took to produce the nystagmus. The normal person shows a rotary nystagmus to the left of good amplitude after 40 seconds douching.

No time must be lost after the nystagmus has been observed before doing the pointing tests. If the examiner is conducting the test alone, he should leave the *recording* of the nystagmus until later. The normal person past-points approximately 8 inches to the right with the right arm and 6 inches to the right with the left arm. This is recorded in the pointing column under "Caloric." By this douching with the head upright, we have tested the vertical canals of the ear douched; the normal nystagmus has demonstrated normal vertical canals and normal pathways from the vertical canals to the eyes. The normal past-pointing has demonstrated, *per se,* a normal vertigo, indicating unobstructed pathways from the vertical canals from the ear douched through the cerebellum to the cerebrum; also normal pathways from the cerebral motor cortex through the brain-stem and cerebellum to the upper extremities.

If the examiner is satisfied that he has obtained a normal past-pointing, he should never repeat the pointing, as he is wasting valuable time. Immediately after taking one pointing of each arm he places the patient's head backward, quickly observes the nystagmus, then again takes the pointing of each arm with the head maintained in this position. With the head 60° back, the right horizontal canal being tested, the normal shows a horizontal nystagmus to the left of good amplitude, and a slightly larger extent of past-pointing than that obtained from the vertical canals with the head upright. These findings are recorded on the chart after the words "Head back."

The left ear is then douched and the nystagmus and past-pointing are recorded in the same way under the respective column, first testing with the head upright and then testing with the head backward 60°.

# CHAPTER XX.

## PRACTICAL CONSIDERATIONS.

Having presented in considerable detail the technic of examination, we are now in a position to examine patients, to discuss the relative value of the different tests and to consider the significance of the phenomena brought out by the examination.

The Nose. If there is any evidence of involvement of the nasal accessory sinuses it must be considered as a possible focal infection, responsible, perhaps, for a toxic labyrinthitis. In the same way empyema of a sinus may be the source of toxemia or may have led to a brain abscess. A deviated septum may be exerting marked pressure on the lateral wall of the nose and may, for example, be the cause of the headache, instead of a suspected intracranial condition. The presence or absence of anesthesia of the nasal mucous membrane may be tested by putting a tubular speculum in the nose to prevent contact with the vestibule and with a delicate cotton-tipped probe, touching various areas within the nose. Naturally only the anterior portions of the nose can as a rule be touched in this way; of course no astringent should be applied to shrink the turbinates, as this would alter the natural tone of the mucous membrane. The special sense of smell is also to be considered; in diseases of the anterior nares odors are lost, whereas in diseases of the posterior nares both odors and flavors are lost. In testing the sense of smell, solutions of different strengths of camphor, rose-water or oil of cloves may be used, and the patient is asked to smell the different solutions and to indicate when he first detects an odor.

The Mouth and Throat. In obscure cases an X-ray of the teeth should be suggested in order to see if there be any pus-pockets at the roots, which not infrequently prove to be the cause of a focal infection. Careful examination of the tonsils in the same way frequently gives evidence of a possible source of systemic infection. In suspected intracranial cases it is of great importance to examine for paralysis or anesthesia. Protrusion of the tongue to the right indicates paresis of the right side of the tongue, showing involvement of the right XII (hypoglossal) nerve. In paralysis of the right side of the velum the uvula is drawn upward toward the unaffected side when the patient says "Ah"; this indicates a paralysis of the IX (glossopharyngeal) Nerve. Examination of the larynx may reveal a paresis or paralysis of the vocal cords; this is very helpful toward diagnosis, showing that there is an involvement of the recurrent laryngeal nerve of that side and, as is well known, this is one of the first of the branches of the X Nerve to become affected. Anesthesia of the mouth, faucial pillars or pharynx indicates an involvement of the IX Nerve. The special sense of taste may be tested by different strength solutions of sugar, quinine or salt.

In a routine examination of the internal ear or in intracranial cases, however, examination of the nose and throat only rarely furnishes important data. On the other hand, the *ear,* of course, needs the most careful and complete study. For example, a patient being studied by a neurologist for a possible intracranial lesion suddenly develops complete deafness in one ear; on examination this proves to be entirely due to a wax-plug in the canal, the removal of which restores the hearing perfectly. Also it has happened in the experience of all otologists that obscure intracranial cases have been cleared up by an ear examination showing an unexpected and latent purulent condition of the ear which has been the cause of a brain abscess or meningitis. On the other hand, many cases complaining of vertigo turn out to be due to acute catarrhal middle ear conditions or Eustachian salpingitis, the relief of which quickly cures the vertigo.

It is well to note the presence of a retraction of the drumhead, because if it is close against the inner wall of the middle ear the effect of cold water is felt by the semicircular canals much more quickly than in the normal ear in which the intervening air space necessitates a longer time before the labyrinth is chilled. In such instances it frequently happens that after douching the ear with cold water, nystagmus will occur after 20 seconds, which is only one-half the usual time required. This is also noted, of course, in cases of perforation of the drum membrane with chronic suppuration in which the cold water comes into intimate relation with the internal ear. On the other hand, in cases of chronic suppuration with thickening of the mucous membrane and perhaps the formation of granulation tissue or polyps, the response is delayed because of the longer time required for the chilling effect of the water to penetrate into the labyrinth. The presence of a perforation of the drum membrane in a case in which there is no active suppuration is a contraindication to douching, as it might possibly start up an inflammatory process. In such cases cold air can be introduced. The actual temperature of the cold air, however, cannot be accurately regulated and a quantitative examination is not possible. Such an examination merely shows whether or not the labyrinth does respond, but it does not allow a determination of how long it took before producing the responses.

## Comparison of Turning and Caloric Tests

(1) In turning, the object is to detect the duration of the nystagmus and the vertigo after turning. In the caloric test, on the other hand, it is noted how long the douching continues before the phenomena develop.

(2) Turning gives much more violent nystagmus, vertigo, past-pointing and falling because (*a*) both ears are necessarily stimulated at the same time; (*b*) the mechanical force of the turning produces a more violent endolymph movement.

(3) The turning stimulus dies quickly whereas the caloric stimulus continues for a long time. The chilling or heating of the wall of the labyrinth produces a continuous current in the endolymph for an average of two minutes; during the continuation of the douching the endolymph movement becomes stronger and stronger and after the douching has stopped the chilled portion of the labyrinth gradually returns to the normal body temperature.

In contrast, the mechanical influence of the momentum produced by the turning is at its height immediately after the chair is stopped and then quickly dies away.

(4) The caloric test affects only the ear douched. This is its chief use. It must be noted also that the vertigo is greater than that produced by turning, because the information from the one ear conflicts with the information from the other ear.

(5) Turning is a sensation to which we are all accustomed. The caloric test produces a disturbance to which we are not accustomed.

(6) Vertigo after turning has the primary and secondary sensations. On turning to the right the patient has the primary sensation of turning to the right, and on stopping he has the secondary sensation of turning to the left. In contrast, after douching there is only a primary sensation due entirely to the douching itself and consisting of a sensation of turning in one direction only.

(7) The position of the head does not affect the subjective sensation of vertigo produced by turning. After the patient is turned to the right he has a sensation of turning to the left whether the head has been inclined backward or forward, if the head is maintained in that position when the chair is stopped. This is so, because of a *previous* experience of turning. After douching, on the other hand, the position of the head has everything to do with the resulting vertigo; douching the right ear with the head back produces exactly the opposite vertigo from douching the right ear with the head forward.

### To Guard Against Unpleasantness to the Patient

The turning-chair should be absolutely steady; its base must not wobble, because the irregularities of movement will obviously tend to make the patient sea-sick; for the same reason the head must be put in the desired position in the head-rest and maintained in that position during the turning.

In performing the caloric test on sensitive people, both the hot and cold water should be ready to use. The sequence of events following the caloric test on neurasthenics or "high-strung" individuals is sometimes as follows: After douching and noting the nystagmus and taking the past-pointing, instead of having the violence of the sensations die away, the patient begins to be nauseated. It is usually possible quickly to note the nystagmus and take the past-pointing and then immediately to douche the same ear with hot water; this will almost invariably stop any unpleasantness. Care must be taken, however, not to continue the douching with the hot water more than perhaps ten or fifteen seconds, as otherwise the hot water itself will begin to produce its own responses. Under these circumstances while douching with the hot water the patient is asked "Do you feel all right now?" In addition the objective signs, nystagmus and falling, may be watched and as soon as they begin to disappear the hot douching is discontinued.

The amount of testing that can be done at one examination should be regulated entirely according to its effect upon that particular patient. Phlegmatic individuals and those whose lesion interrupts the normal responses frequently permit the examiner to complete all the tests at one sitting. The average case, however, had best be examined on two or three separate occasions. Our method is, on the first examination to take notes of the chief complaint and the history, examine the nose, throat and ears, take the tuning-fork tests and examine for spontaneous nystagmus, spontaneous pointing and the various falling tests. There is nothing in this series of examinations to give the slightest alarm to the most nervous patient. Perhaps also on the first day we may examine for nystagmus after turning; this gives the patient some little knowledge of what the chair is for and at the same time reassures him that it is not anything to be dreaded. The next day the past-pointing after turning is undertaken and then the douching of one ear. The douching of the other ear may be done on the second examination if the other tests have not proven unpleasant to the patient or may be postponed till the third examination. On the average, two examinations on two different days are sufficient to fill in the chart, but it is far better to take three, four or even five days before completing the chart, rather than antagonize a nervous patient by making the tests unpleasant to him. Of course this is entirely a matter of studying the disposition and idiosyncracies of the patient. In some cases, highly neurotic, ignorant or difficult to manage, it is best to do the caloric test first of all so that if the patient refuses further examination we will have performed the most useful of the tests. With a little care in these particulars, however, it will be found that not one patient in twenty will complain of any unpleasantness whatever.

If the patient is too sick to get out of bed, the ears can be douched very satisfactorily with the patient lying on his back; the pillow naturally raises the head slightly and puts the horizontal canals therefore in an exactly vertical position. The pointing can be undertaken very easily by having the patient feel the examiner's finger at a point alongside of the patient's thigh and then raise his arm to the vertical position and come back again.

## VALUE OF SUMMARIES

During the examination, as we record the findings on the chart, it is very helpful to *draw conclusions* as we go along step by step—to "face the issue" on each test; for example, after examining for spontaneous nystagmus or spontaneous pointing it is best to write down immediately what these tests themselves indicate. Again, if after turning to the right there is a horizontal nystagmus to the left lasting 16 seconds, and after turning to the left there is a horizontal nystagmus to the right of 8 seconds, we then write immediately "Suggests impaired right horizontal canal." This is exactly the reaction that would take place if the left horizontal canal alone were functionating; we therefore write down the immediate conclusions from this test itself. In a similar way if after douching the right ear with the head upright, nystagmus does not appear for perhaps a minute or more, or if the amplitude is poor, we write "Impaired right vertical canals." If after turning or douching there is an absence of past-pointing, we write immediately under each separate test what that failure of reaction would indicate. Then after all the tests are completed we have a series of separate conclusions from which the final conclusion can be drawn.

After the completion of all the tests it is important to put down at once the logical deduction from the examinations, paying no attention at all as to whether it seems to fit in with a common-sense view of the case. If an apparently normal person shows certain deviations from the normal it is best to write down for example, "Suggests lesion of the right cerebellar hemisphere," even though one should feel practically sure that the patient had no lesion of the cerebellum whatever. The next examination may show that it was entirely a chance deviation and the conclusion can then be altered accordingly; on the other hand, if the deviations persist, the old conclusion should be adhered to. In certain cases of this type in our experience, time has demonstrated a cerebellar lesion. It is essential that the ear-tests should be conducted for themselves alone, disregarding what we may know of others' opinions of the case and confining the report entirely to the conclusions suggested *purely from the ear viewpoint.*

## METHODS OF REMEMBERING NORMAL RESPONSES FROM THE TESTS

The usual routine methods of examination are—(1) Turning, with the head upright, and (2) douching, with the head upright. If to the RIGHT (*i.e.,* either turning to the right or douching the right ear) everything is to the RIGHT—all the pointing, shoulder from above, shoulder from below, elbow, wrist, neck, trunk and also the falling—all are to the RIGHT. The deviation of the eyes, just as all the other responses, is also to the RIGHT. This, however, is the *slow* movement of the eyes to the right; the "recovery," the *quick* component to the left, is more conspicuous and therefore this phenomenon is termed "Nystagmus to the left." The vestibular pull of the eyes, however, is just as truly to the right as all the other responses. *Vice versa,* to emphasize this; if the LEFT ear is douched or the patient turned to the left, all the responses are to the LEFT; the true vestibular movement of the eyes is also to the left although we *speak* of it as "Nystagmus to the right." In routine examinations, therefore, it is helpful merely to remember

> If to the right, everything is to the right.
> If to the left, everything is to the left.

This method of remembering the responses is useful only to the beginner; after we are familiar with the fundamental physiology of the labyrinth, and recognize in what direction the endolymph is moving after any given test, these suggestions are no longer necessary. We simply note that the vestibular pull of the eyes, the past-pointing and the falling all occur in the direction of the endolymph movement.

# CHAPTER XXI

## PATHOLOGIC CONSIDERATIONS

It will be observed that up to this point we have dealt only with the normal hearing tests and the normal responses to the tests of the vestibular apparatus. We now discuss the abnormal. We have considered only the anatomy and physiology of the parts involved, the tracts connecting them, and the methods of examination of these tracts, to determine whether they are intact or not. We are now concerned with the interpretation of pathologic findings—the significance of deviations from the normal.

### Interpretation of the Findings in the Auditory Mechanism

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 0 | A | 4 | Ac < Bc > n | $C_4$ | good | Galt. | .7 | Pol. | — | Weber |
| | 3 | | 8 | Ac > Bc = n | | good | | .7 | | + | |

indicate *catarrhal* deafness in the right ear. Left ear normal. In the right ear the low fork is not heard at all and the middle fork is reduced one-half; this is characteristic of obstructive lesions of the middle or external ear—the low sounds are lost before the higher sounds. The high tones of the C4 fork and the upper limit of the Galton whistle are heard perfectly, showing that the cochlea is intact for the upper tones. Air-conduction is greater than Bone-conduction in the normal, and also in a lesion of the internal ear. Here Ac Bc shows an obstructing lesion of the conducting apparatus. Also the fact that the Bone-conduction is greater than normal, shows that the internal ear is intact but there is an obstruction in the conducting apparatus. The Politzer test being negative shows an obstruction of the right eustachean tube. The Weber test being lateralized to the right, shows a lesion of the conducting apparatus of the right ear. The cochlea and VIII Nerve are evidently normal, as the high tones are heard perfectly and the Bone-conduction is greater than normal.

The following hearing tests

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| A<br>= | 3 | A | 4 | Ac > Bc < n | $C_4$ | poor | Galt. | 3.4 | Pol. | + |
| | 3 | | 8 | Ac > Bc = n | | good | | .7 | | +Weber |

indicate impairment of the auditory fibres of the right ear, either in the labyrinth or VIII Nerve. The low tones are only moderately impaired, whereas the high tones are markedly diminished; this is characteristic of nerve-lesion, in that the low tones are usually the last to be lost and the high tones are the first to go. Bone-conduction less than normal and the Weber lateralized to the normal side, both show involvement of the receptive apparatus.

The following hearing tests

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| A<br>= | o | A | o | Ac? Bc? n | $C_4$ | none | Galt. | none | Pol. | − |
| | 3 | | 8 | Ac > Bc = n | | good | | .7 | | +Weber |

indicate complete destruction of the auditory fibres in either the cochlea or VIII Nerve of the right ear. To corroborate this diagnosis it will be found that the 200 fork placed upon the right mastoid process will be heard only in the left ear, showing that the internal ear on the right side is completely destroyed. In order to settle the question finally we can exclude the left, or normal ear, and then test the right ear. This can be done as follows:

(1) A noise-apparatus, such as the Barany model, is placed in the normal ear; the peculiar noise produced completely engrosses the internal ear of that side, in this way eliminating this ear. Shouting in the other ear, or any other noise whatsoever, is not heard by the patient. A vibrating tuning-fork placed at any point on the head is not heard. These tests have shown that Air-conduction and Bone-conduction are both completely lost in the affected ear.

(2) During the examination of the ear with the pneumatic otoscope, while the drum membrane is being moved in and out by pressure and suction, this ear is unable to hear any sound by Air-conduction; so that any shouting that is understood by the patient is being heard by the affected ear.

(3) Later, during the vestibular tests, we eliminate the normal ear in a similar way by douching; douching very satisfactorily shuts off sounds from the ear, not only by the presence in the external canal of the water and its impact upon the drumhead, but also because the actual noise itself produced by the douching is sufficient to eliminate that ear.

These functional tests of hearing therefore have made a differential diagnosis between an *obstructive* lesion of the external or middle ear and a *receptive* lesion of either the internal ear or VIII Nerve.

As to the intracranial paths of the auditory fibres, they unfortunately give us very little information of value in locating lesions. Randall suggests, however, that a lesion involving the trapezoid bodies in the pons would be capable of causing complete binaural deafness; his reason being that not only the anterior fibres enter the trapezoid bodies but that also the posterior fibres from the acoustic striæ come forward to the trapezoid body. A lesion involving both these bodies or the fibres between them would, according to this view, block all auditory impulses from both ears.

"Word-deafness" consists of inability to understand the meaning of spoken words. In such a case the tuning-fork tests may show perfect hearing for *tone;* the spoken voice also is heard, but the words are as *unintelligible* as if they were in a *foreign language.* This indicates a lesion of the cerebral cortical centre for word-hearing, which is generally regarded to be located in the posterior part of the first and the adjoining superior portion of the second temporal convolutions. "Word-deafness" in the right ear is suggestive of a lesion of the *left* cerebral cortex, and *vice versa;* this, however, is by no means absolute, as unquestionably some auditory fibres go to the cerebral cortex of the same side.

## INTERPRETATION OF FINDINGS IN THE VESTIBULAR MECHANISM

On looking to the right or looking to the left there is often a slight nystagmus, due to a wavering in the pull of the muscles in this extreme position; this is physiologic. When there is a marked nystagmus or a noticeable difference between looking to the right or looking to the left, it should be considered pathologic. A spontaneous nystagmus of any type or in any direction which is present on looking straight ahead, is always pathologic. A spontaneous vertical nystagmus, on looking straight ahead, or looking up or looking down, is indicative of a lesion involving the brain-stem. A lesion of the labyrinth or VIII Nerve never produces a spontaneous vertical nystagmus.

Spontaneous Vertigo. A continuous vertigo lasting over three months is strongly suggestive either of an intracranial lesion or neurasthenia. Vertigo due to a lesion of the labyrinth passes away as a rule in a few days or weeks.

Spontaneous Pointing. The patient occasionally, through carelessness or inattention, will show a spontaneous past-pointing; several repetitions, however, will prove that this past-pointing was not pathologic, but merely accidental. A spontaneous past-pointing of the right arm to the right, suggests a lesion of the inward-pointing centre of the right cerebellar hemisphere. In an attempt to find the finger, there is lacking the restraining influence of the inward-pointing centre and the past-pointing is therefore outward. In a similar way a spontaneous past-pointing of the right arm to the left suggests a lesion of the outward-pointing centre of the right cerebellar hemisphere. It is usually sufficient to examine only the upper extremities; however, if the patient exhibits difficulty in walking or standing, the lower extremities should also be tested. In obscure cases all the tests may be carried out in order to examine all the cerebellar cortical centres.

Spontaneous Falling. A patient with a lesion of the right labyrinth tends to fall in the direction of the right ear; when he faces forward he tends to fall to the right, on turning the head to the right he tends to fall backwards and on turning the head to the left he tends to fall forward. This, as a rule, is not true of an intracranial lesion; if the lesion is on the right side the patient tends to fall to the right, and if the lesion is on the left side the patient tends to fall to the left, regardless of the position of the head. In the attempt to overthrow the patient, we discover whether the pelvic girdle shows a normal swaying to the opposite side in order to preserve equilibrium; if the pelvic girdle fails to respond, it suggests a lesion of the vermis of the cerebellum in the area presiding over the pelvic girdle.

### FAILURE OF REACTION TO EAR-STIMULATION

It is the absence of normal responses or the deviation from the normal responses that gives us information as to a lesion along the path of those particular fibres that fail to transmit the impulses (see Chapter VI.) Speaking in general, if after stimulation of the ears there is no nystagmus, a lesion is indicated along the vestibulo-ocular tract which should normally have produced nystagmus. If there is no vertigo there is a lesion in the course of the vestibulo-cerebello-cerebral tract, which normally conveys to the cerebrum the impulses producing vertigo. If there is no past-pointing after ear-stimulation, it shows either

(1) A lesion along the vestibulo-cerebello-cerebral tract just mentioned; in this case the absence of vertigo naturally causes an absence of past-pointing. It must be noted, however, that it causes an absence of past-pointing of all extremities in both directions; as there is no subjective sensation of turning, there is no attempt of any extremity to past-point.

(2) A lesion along the pointing tracts themselves, either in the cerebrum, the cerebellar peduncles, the cerebellar nuclei or the cerebellar cortex. It is to be noted that a lesion under these circumstances would involve only that particular pointing affected by the fibres involved. For example, a lesion of the right cerebellar hemisphere in the region of the outward-pointing centre, would show a failure of the right arm to past-point to the right, when it should past-point to the right; the right arm would past-point properly to the left and the left arm would past-point properly both to the right and to the left. This makes a sharp contrast to the lesion mentioned above in which, owing to the absence of *vertigo,* there was a complete absence of past-pointing of all extremities in both directions.

The absence of falling after ear-stimulation similarly indicates a lesion either of some portion of the vestibulo-cerebello-cerebral tract or of the cerebro-cerebello-spinal tract.

Either a "perverted" nystagmus or an "inverse" nystagmus is indicative of involvement of the brain-stem. If a given ear-stimulation should normally produce a horizontal nystagmus to the left, and under these circumstances there appears instead a vertical nystagmus upwards, a rotary nystagmus, or an oblique nystagmus, we may speak of it as a "perverted" nystagmus; if instead of the normal horizontal nystagmus to the left there occurs instead a horizontal nystagmus to the *right,* it may be termed an "inverse" nystagmus. It is evident that under these circumstances the impulses from the ear have been conducted to the *wrong* nuclei. Neither a perverted nor an inverse nystagmus can possibly be produced by a lesion of the labyrinth or VIII Nerve; a peripheral lesion produces a poor nystagmus or no nystagmus at all, but an absolutely false response would of necessity indicate involvement of the brain-stem.

If after ear-stimulation there occurs a conjugate deviation of the eyes instead of a nystagmus, this absence of the quick component of the nystagmus indicates a lesion at the base of the cerebral crura where the fibres from the cerebrum are distributed to the eye-muscle nuclei or at some point higher up along the cerebro-ocular tracts.

The foregoing shows in general the method of analysis on the basis of the auditory and vestibular tracts. These methods will be presented in more detail in the two following chapters.

# CHAPTER XXII

## HYPOTHETICAL CASES

A STUDY of the differential diagnosis between lesions of the internal ear and of the intracephalic structures, as well as the general subject of intracranial localization, furnishes so much data that the analysis of any given pathologic case may appear very complex to those taking up this work for the first time. Therefore, in order to bring out the fundamental principles in the analysis of these pathologic cases, it will prove helpful first to take up hypothetical cases, putting down in dogmatic form the phenomena to be expected from a lesion in any given location.

The method of approach is based upon the tracts previously presented. For example, the fibres from the horizontal semicircular canal proceed to Deiters' nucleus and divide at this point; given an isolated lesion between Deiters' nucleus and the posterior longitudinal bundle, all responses from all the semicircular canals would be normal *except* that the right horizontal canal would fail to produce *nystagmus.* Similarly, given a lesion limited to the right inferior cerebellar peduncle, all responses from all semicircular canals would be normal *except* that the right horizontal canal would fail to produce *vertigo.*

Of course, in actual pathologic cases, we usually find that more than one response is absent or impaired, and we also may find the added difficulty of the effect of intracranial pressure upon some of the pathways. If, however, we first master these hypothetical cases, we are then in a position to approach the study of the actual cases given in the final chapter.

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## CHART III A

Name Age Date
Address
Referred by

DIAGNOSIS: Recent destruction of Right labyrinth

SUMMARY: Normal responses from Left labyrinth. No responses whatever from Right labyrinth. Stone deaf in Right ear.

Complains of Vertigo, staggering, recent deafness, Right side.
Recent tinnitus, Right side.

HISTORY:
Dizziness Yes, marked.
Staggering Yes, marked
Deafness Total in Right
Tinnitus At time of attack in Right ear, then ceased.

NOSE: Negative

THROAT: Negative

EARS:
A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 0 | A | 0 | Ac ? Bc ? n | Pol. | — | $c^4$ | 0 | Galt | 0 | Weber |
| | 3 | | 8 | Ac > Bc = n | | + | | Good | | .7 | To Left. |

## CHART III B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT Marked → ↷
Looking UP None
Looking DOWN None

POINTING

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 2" to Right. | 2" to Right |

Nystagmus on looking to Left
Vertigo present
Past-pointing to Right, both arms
Falling to Right
Romberg to Right
Turning head to right falls backward
Turning head to left falls forward
Attempt to overthrow pelvic girdle, normal reactions

---

### TURNING

To RIGHT →
Amp. Good
Duration 16 Sec.

| | | |
|---|---|---|
| To RIGHT<br>Shoulder from above | 8" to Right | 6" to Right |

Nystagmus impaired duration, but good amplitude.
Vertigo 2/3 of normal (16 seconds)
Past-pointing 2/3 of normal, but correct direction

To LEFT ←
Amp. Good
Duration 8 Sec.

| | | |
|---|---|---|
| To LEFT<br>Shoulder from above | 3" to Left | 4" to Left |

Nystagmus shortened
Vertigo 1/3 of normal (8 seconds)
Past-pointing 1/3 of normal, but correct direction

---

### CALORIC

Douche RIGHT None
Amp.
After 5 min. sec.

| | | |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | 2" to Right | 2" to Right |

Same as spontaneous

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back None
Amp.

2" to Right. 2" to Right.
Same as spontaneous.

Douche LEFT ↶
Amp. Good
After min. 40 sec.

| | | |
|---|---|---|
| Douche LEFT<br>Shoulder from above | 6" to Left | 8" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back ←
Amp. Good

8" to Left 8" to Left

From Dr. I. H. Jones' Equilibrium and Vertigo.

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## CHART IV A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion between the Right Deiters' Nucleus and Posterior Longitudinal Bundle

SUMMARY: All responses, nystagmus, vertigo, past-pointing and falling are normal except that Right horizontal canal fails to produce nystagmus.

Complains of Twitching of eyes.

HISTORY:
Dizziness No
Staggering No
Deafness No
Tinnitus No.

NOSE: Negative

THROAT: Negative

EARS: A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | Weber |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3 | A | 8 | Ac > Bc = n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
| | 3 | | 8 | Ac > Bc = n | | + | | Good | | .7 | + |

## CHART IV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS
Looking to RIGHT None
Looking to LEFT Marked →
Looking UP None
Looking DOWN None

POINTING RIGHT | LEFT

Shoulder from above Touched | Touched

Nystagmus on looking to Left.
Vertigo None
Past-pointing None
Falling None
Romberg Negative
Turning head to right Negative
Turning head to left Negative
Attempt to overthrow Normal reactions.

### TURNING

To RIGHT →
Amp. Good
Duration 16 Sec.

To RIGHT
Shoulder from above 12" to Right | 10" to Right

Nystagmus shortened duration
Vertigo Normal
Past-pointing Normal

To LEFT ←
Amp. Good
Duration 8 Sec.

To LEFT
Shoulder from above 10" to Left | 12" to Left

Nystagmus shortened duration
Vertigo Normal
Past-pointing Normal

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 40 sec.

Douche RIGHT
Shoulder from above 8" to Right | 6" to Right

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back No nystagmus Normal vertigo 10" to Right | 8" to Right
Amp.

Douche LEFT ↶
Amp. Good
After min. 40 sec.

Douche LEFT
Shoulder from above 6" to Left | 8" to Left

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back ← 8" to Left | 10" to Left
Amp. Good

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## CHART V A

Name Age Date
Address
Referred by

**DIAGNOSIS:** Lesion Right side of Pons, posterior aspect, next to but not involving the posterior longitudinal bundle.

**SUMMARY:** All responses normal, nystagmus, vertigo, past-pointing and falling from all canals *except* that Right vertical canals fail to produce nystagmus.

Complains of Twitching of eyes.

**HISTORY:**
Dizziness No
Staggering No.
Deafness No
Tinnitus No

**NOSE:** Negative

**THROAT:** Negative

**EARS:**
A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

|  |  |  |  |  |  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  |  |  |  |  |  |  |  |  | Weber |
| A | 3 | A | 8 | Ac > Bc = n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
|  | 3 |  | 8 | Ac > Bc = n |  | + |  | Good |  | .7 | − |

## CHART V B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None

Looking to LEFT ↷

Looking UP ↑ may or may not be present

Looking DOWN ↓ may or may not be present

POINTING

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus on looking to Left
Vertigo None
Past-pointing None
Falling None
Romberg None
Turning head to right Negative
Turning head to left Negative
Attempt to overthrow Normal pelvic girdle reactions.

---

### TURNING

To RIGHT →
Amp. Good
Duration 24 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 12". to Right | 10" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

To LEFT ←
Amp. Good
Duration 24 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 10" to Left | 12" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

---

### CALORIC

Douche RIGHT None
Amp.
After 5 min. sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 8" to Right | 6" to Right |

Nystagmus None
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back →
Amp. Good

Normal vertigo.

| | RIGHT | LEFT |
|---|---|---|
| Head Back | 10" to Right | 8" to Right |

Douche LEFT ↶
Amp. Good
After min. 40 sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 6" to Left | 8" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back ←
Amp. Good

| | RIGHT | LEFT |
|---|---|---|
| Head Back | 8" to Left | 10" to Left. |

From Dr. I. H. Jones' Equilibrium and Vertigo

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## CHART VI A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion Right side of pons, in lower third near median line

SUMMARY Right vertical canals fail to produce any responses. All other canals produce the normal nystagmus, vertigo, past-pointing and falling.

Complains of Vertigo, staggering and twitching of eyes

HISTORY:
Dizziness Yes
Staggering Yes
Deafness No
Tinnitus No

NOSE: Negative

THROAT: Negative

EARS:
A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3 | A | 8 | Ac | > | Bc | = | n | Pol. | + | $c^4$ Good | Galt | .7 |
| – | 3 | | 8 | Ac | > | Bc | = | n | | + | Good | | .7 |

## CHART VI B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT ↷
Looking UP ↑ may or may not be present
Looking DOWN ↓ may or may not be present.

**POINTING**

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 1" to Right | 1" to Right. |

Nystagmus on looking to Left.
Vertigo Yes
Past-pointing to Right, both arms
Falling to Right.
Romberg falls to Right.
Turning head to right falls to Right
Turning head to left falls to Right
Attempt to overthrow normal pelvic girdle reactions.

---

### TURNING

To RIGHT →
Amp. Good
Duration 24 Sec.

| | | |
|---|---|---|
| To RIGHT<br>Shoulder from above | 13" to Right | 11" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

To LEFT ←
Amp. Good
Duration 24 Sec.

| | | |
|---|---|---|
| To LEFT<br>Shoulder from above | 9" to Left | 11" to Left. |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

---

### CALORIC

Douche RIGHT None
Amp.
After 5 min. sec.

| | | |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | 1" to Right | 1" to Right |

Same as spontaneous.

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back →
Amp. Good

normal vertigo. 11" to Right 9" to Right

Douche LEFT ↶
Amp. Good
After min. 40 sec.

| | | |
|---|---|---|
| Douche LEFT<br>Shoulder from above | 5" to Left | 7" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back ←
Amp. Good

7" to Left 9" to Left

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## CHART VII A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion of Right Inferior Cerebellar Peduncle.

SUMMARY: All phenomena normal, nystagmus, vertigo, past-pointing and falling, from all canals, except that Right horizontal canal fails to produce vertigo, and its consequent past-pointing.

Complains of Vertigo

HISTORY:
Dizziness Yes
Staggering Yes
Deafness No
Tinnitus No

NOSE: Negative

THROAT: Negative

EARS:
A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | Weber. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3 | A | 8 | Ac > Bc = n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
| = | 3 | | 8 | Ac > Bc = n | | + | | Good | | .7 | − |

## CHART VII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None.

POINTING

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 1" to Right | 1" to Right |

Nystagmus None
Vertigo Yes
Past-pointing Slight to Right, both arms
Falling to Right.
Romberg falls to Right.
Turning head to right falls to Right
Turning head to left falls to Right
Attempt to overthrow pelvic girdle reactions normal.

---

### TURNING

To RIGHT →
Amp. Good
Duration 24 Sec.

To RIGHT
Shoulder from above 8" to Right 6" to Right.

Nystagmus Normal
Vertigo 2/3 of normal (16 seconds.)
Past-pointing 2/3 of normal but correct direction

To LEFT ←
Amp. Good
Duration 24 Sec.

To LEFT
Shoulder from above 3" to Left 4" to Left.

Nystagmus Normal
Vertigo 1/3 of normal (8 seconds.)
Past-pointing 1/3 of normal, but correct direction.

---

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 40 sec.

Douche RIGHT
Shoulder from above 9" to Right 7" to Right

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back →
Amp. Good

1" to Right 1" to Right.
Same as spontaneous

Douche LEFT ↶
Amp. Good
After min. 40 sec.

Douche LEFT
Shoulder from above 5" to Left 7" to Left.

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back ←
Amp. Good

7" to Left 9" to Left

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## CHART VIII A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion of Right middle cerebellar peduncle.

SUMMARY. All responses normal, nystagmus, vertigo, past-pointing and falling, from all the canals *except* that Right vertical canals fail to produce vertigo and its consequent past-pointing and falling

Complains of Vertigo and staggering.

HISTORY:
Dizziness Yes
Staggering Yes
Deafness No
Tinnitus No

NOSE: Negative

THROAT: Negative

EARS: A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | | | | | Weber |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3 | A | 8 | Ac | > | Bc | = | n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
| = | 3 | | 8 | Ac | > | Bc | = | n | | + | | Good | | .7 | − |

# CHART VIII B

## TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 1" to Right | 1" to Right |

Nystagmus None
Vertigo Yes
Past-pointing to Right both arms
Falling to Right
Romberg to Right
Turning head to right does not change direction of falling
Turning head to left " " " " " ".
Attempt to overthrow normal pelvic girdle reactions

---

### TURNING

To RIGHT →
Amp. Good
Duration 24 Sec.

| To RIGHT | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 13" to Right | 11" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

To LEFT ←
Amp. Good
Duration 24 Sec.

| To LEFT | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 9" to Left | 11" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

---

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 40 sec.

| Douche RIGHT | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 1" to Right | 1" to Right |

Same as spontaneous.

Nystagmus Normal
Vertigo None
Past-pointing None
Falling None

Head Back →
Amp. Good

| Head Back | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| | 11" to Right | 9" to Right |

Douche LEFT ↶
Amp. Good
After min. 40 sec.

| Douche LEFT | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 5" to Left | 7" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back ←
Amp. Good

| Head Back | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| | 7" to Left | 9" to Left. |

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## CHART IX A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion of Right superior cerebellar peduncle

SUMMARY: Right horizontal and Right vertical canals fail to produce vertigo and its consequent past-pointing and falling; they produce normal nystagmus.
All responses from Left ear normal.

Complains of Vertigo, staggering.

HISTORY:
Dizziness Yes
Staggering Yes
Deafness No
Tinnitus No.

NOSE: Negative

THROAT: Negative

EARS: A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | Weber |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3 | A | 8 | Ac > Bc = n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
| = | 3 | | 8 | Ac > Bc = n | | + | | Good | | .7 | — |

## CHART IX B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

**Looking to RIGHT** None
**Looking to LEFT** None
**Looking UP** None
**Looking DOWN** None

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 2" to Right | 2" to Right |

Nystagmus None.
Vertigo Yes
Past-pointing both arms to Right
Falling to Right
Romberg falls to Right
Turning head to right does not alter direction of falling.
Turning head to left " " " " " .
Attempt to overthrow normal pelvic girdle reactions.

---

### TURNING

**To RIGHT** →
Amp. Good
Duration 24 Sec

| | RIGHT | LEFT |
|---|---|---|
| To RIGHT<br>Shoulder from above | 8" to Right | 6" to Right |

Nystagmus Normal
Vertigo 2/3 of normal (16 seconds)
Past-pointing 2/3 of normal but correct direction.

**To LEFT** ←
Amp. Good
Duration 24 Sec.

| | RIGHT | LEFT |
|---|---|---|
| To LEFT<br>Shoulder from above | 3" to Left | 4" to Left |

Nystagmus Normal
Vertigo 1/3 of normal (8 seconds)
Past-pointing 1/3 of normal but correct direction.

---

### CALORIC

**Douche RIGHT** ↷
Amp. Good
After min. 40 sec.

| | RIGHT | LEFT |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | 2" to Right | 2" to Right |

Same as spontaneous.

Nystagmus Normal
Vertigo None
Past-pointing None
Falling None

**Head Back** →
Amp. Good

| RIGHT | LEFT |
|---|---|
| 2" to Right | 2" to Right |

Same as spontaneous

**Douche LEFT** ↶
Amp. Good
After min. 40 sec.

| | RIGHT | LEFT |
|---|---|---|
| Douche LEFT<br>Shoulder from above | 4" to Left | 6" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal.

**Head Back** ←
Amp. Good

| RIGHT | LEFT |
|---|---|
| 6" to Left | 8" to Left. |

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## CHART X A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion of outward pointing centre for the Right arm, of Right cerebellar hemisphere

SUMMARY: All phenomena normal, nystagmus, vertigo, past-pointing and falling, except that Right arm cannot be made to past-point to Right.

Complains of Lack of control of Right upper extremity

HISTORY:
Dizziness No
Staggering No
Deafness No
Tinnitus No

NOSE: Negative

THROAT: Negative

EARS:
A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | Weber |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3 | A | 8 | Ac > Bc = n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
| = | 3 | | 8 | Ac > Bc = n | | + | | Good | | .7 | − |

## CHART X B

### TESTS OF THE VESTIBULAR APPARATUS

#### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT *None*
Looking to LEFT *None*
Looking UP *None*
Looking DOWN *None*

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | *3" to Left* | *Touched* |

Nystagmus *None*
Vertigo *None*
Past-pointing *Right arm to Left*
Falling *None*
Romberg *falls to Right*
Turning head to right *does not change direction of falling*
Turning head to left *" " " " ".*
Attempt to overthrow *normal pelvic girdle reactions.*

#### TURNING

To RIGHT →
Amp. *Good*
Duration *24* Sec.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| To RIGHT<br>Shoulder from above | *Touched* | *10"* to *Right* |

Nystagmus *Normal*
Vertigo *Normal*
Past-pointing *absent of Right arm to Right*

To LEFT ←
Amp. *Good*
Duration *24* Sec.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| To LEFT<br>Shoulder from above | *13"* to *Left* | *12"* to *Left.* |

Nystagmus *Normal*
Vertigo *Normal*
Past-pointing *Normal*

#### CALORIC

Douche RIGHT ↷
Amp. *Good*
After min. *40* sec.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | *Same as spontaneous*<br>*3"* to *Left* | *6"* to *Right* |

Nystagmus *Normal*
Vertigo *Normal*
Past-pointing *absent of Right arm to Right.*
Falling *Normal*

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Head Back →<br>Amp. *Good* | *Same as spontaneous*<br>*3"* to *Left* | *8"* to *Right* |

Douche LEFT ↶
Amp. *Good*
After min. *40* sec.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Douche LEFT<br>Shoulder from above | *9"* to *Left* | *8"* to *Left* |

Nystagmus *Normal*
Vertigo *Normal*
Past-pointing *Normal*
Falling *Normal*

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Head Back ←<br>Amp. *Good* | *11"* to *Left* | *10"* to *Left* |

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## CHART XI A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion of inward pointing centre of Right arm, in Right cerebellar hemisphere

SUMMARY: All phenomena normal, nystagmus, vertigo, past-pointing and falling, *except* the Right arm cannot be made to point to Left

Complains of Lack of control of Right upper extremity

HISTORY:
Dizziness No
Staggering No
Deafness No
Tinnitus No

NOSE: Negative

THROAT: Negative

EARS: A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | Weber |
| $\underline{A}$ | 3 | A | 8 | Ac > Bc = n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
| = | 3 | | 8 | Ac > Bc = n | | + | | Good | | .7 | − |

## CHART XI B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT None
Looking UP
Looking DOWN

| POINTING | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 3" to Right | Touched |

Nystagmus None
Vertigo None
Past-pointing Right arm to Right
Falling None
Romberg Negative
Turning head to right Negative
Turning head to left Negative
Attempt to overthrow normal pelvic girdle reactions

### TURNING

To RIGHT →
Amp. Good
Duration 24 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 12" to Right | 10" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

To LEFT ←
Amp. Good
Duration 24 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 3" to Right | 12" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Absent of Right arm to Left

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 40 sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 8" to Right | 6" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | RIGHT | LEFT |
|---|---|---|
| Head Back →<br>Amp. Good | 10" to Right | 8" to Right |

Douche LEFT ↶
Amp. Good
After min. 40 sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 3" to Right | 8" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Absent of Right arm to Left
Falling Normal

| | RIGHT | LEFT |
|---|---|---|
| Head Back ←<br>Amp. Good | 3" to Right | 10" to Left |

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## CHART XII A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion of Right-side of vermis of the cerebellum.

SUMMARY: Nystagmus, vertigo and past-pointing of arms, after stimulation, normal. There is a spontaneous falling to the Right and no increased tendency of falling to the Right after such stimulation as should produce a falling to the Right

Complains of Loss of control of trunk

HISTORY:
Dizziness No
Staggering Yes
Deafness No
Tinnitus No

NOSE: Negative

THROAT: Negative

EARS: A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | Weber |
| A | 5 | A | 8 | Ac > Bc = n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
| = | 3 | | 8 | Ac > Bc = n | | + | | Good | | .7 | — |

## CHART XII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS
Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

POINTING

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling falls to Right
Romberg falls to Right
Turning head to right falls to Right
Turning head to left falls to Right
Attempt to overthrow pelvic girdle fails to bend to Right when patient is pushed toward Left

### TURNING

To RIGHT →
Amp. Good
Duration 24 Sec.

To RIGHT
Shoulder from above 12" to Right 10" to Right

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Trunk from below fails to past-point to Right

To LEFT ←
Amp. Good
Duration 24 Sec.

To LEFT
Shoulder from above 10" to Left 12" to Left

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Trunk from below past-points to Left

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 40 sec.

Head Back →
Amp. Good

Douche RIGHT
Shoulder from above 8" to Right 6" to Right

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Absence of increased falling to Right as compared to spontaneous
Trunk from below fails to past-point to Right 10" to Right 8" to Right

Douche LEFT ↶
Amp. Good
After min. 40 sec.

Head Back ←
Amp. Good

Douche LEFT
Shoulder from above 6" to Left 8" to Left

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal falling to Left
Trunk from below past-points to Left 8" to Left 10" to Left

From Dr. I. H. Jones' Equilibrium and Vertigo

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8✓

## CHART XIII A

Name Age Date
Address
Referred by

DIAGNOSIS: Lesion at decussation of superior cerebellar peduncles at base of cerebral crura

SUMMARY: 1. All canals, both ears, fail to produce vertigo, and its consequent past-pointing and falling
2 Instead of a nystagmus, there is produced a conjugate deviation, because of involvement at this point of the cerebral fibres to the eye-muscle nuclei.

Complains of Vertigo and staggering

HISTORY:
Dizziness Yes
Staggering Yes
Deafness no
Tinnitus no

NOSE: Negative

THROAT: Negative

EARS: A. D. Negative
A. S. Negative

Fistula

Hearing Tests

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | | | Weber |
| A | 3 | A | 8 | Ac | > | Bc | = | n | Pol. | + | $c^4$ | Good | Galt | .7 | + |
| = | 3 | | 8 | Ac | > | Bc | = | n | | + | | Good | | .7 | − |

## CHART XIII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

POINTING

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus None
Vertigo present
Past-pointing none
Falling None
Romberg
Turning head to right negative
Turning head to left negative
Attempt to overthrow normal pelvic girdle reactions.

---

### TURNING

To RIGHT
Amp.
Duration 24 Sec.
No nystagmus but a conjugate deviation to Right

To RIGHT
Shoulder from above Touched to Touched
Nystagmus None, but a conjugate deviation
Vertigo None
Past-pointing None

To LEFT
Amp.
Duration 24 Sec.
No nystagmus but a conjugate deviation to Left

To LEFT
Shoulder from above Touched to Touched
Nystagmus None, but a conjugate deviation
Vertigo None
Past-pointing None

---

### CALORIC

Douche RIGHT
Amp.
After min. sec.
No nystagmus but a conjugate rolling of eyes to Right

Douche RIGHT
Shoulder from above Touched to Touched
Nystagmus None, but a conjugate rolling.
Vertigo None
Past-pointing None
Falling None
Head Back Conjugate deviation to Right Touched to Touched
Amp.

Douche LEFT
Amp.
After min. sec.
No nystagmus but a conjugate rolling of eyes to Left

Douche LEFT
Shoulder from above Touched to Touched
Nystagmus None, but a conjugate rolling to Left
Vertigo None
Past-pointing None
Falling None
Head Back Conjugate deviation to Left Touched to Touched
Amp.

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a√

## CHART XIV A

Name Age Date
Address
Referred by

DIAGNOSIS: Tumor of Right cerebello-pontile angle

SUMMARY: Stone deaf Right ear. Right static labyrinth fails in all responses. Left vertical canals fail in all responses, whereas Left horizontal gives normal responses.

Complains of Vertigo, staggering, deafness in Right ear, headache.

HISTORY:
Dizziness Yes
Staggering Yes
Deafness Yes, in Right ear
Tinnitus History of tinnitus in Right ear

NOSE: Negative

THROAT: Negative

EARS:
A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests ·

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 0 | A | 0 | Ac | ? | Bc | ? | n | Pol. | — | $c^4$ | Galt | 0 | Weber |
| = | 3 | | 8 | Ac | > | Bc | = | n | | + | | | .7 | to Left |

## CHART XIV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

POINTING
RIGHT LEFT

Looking to RIGHT None
Shoulder from above Touched Touched

Looking to LEFT marked → ↷
Looking UP ↑
Looking DOWN ↓

Nystagmus on looking to Left and on looking upward and downward
Vertigo Yes
Past-pointing None
Falling to Right
Romberg falls to Right
Turning head to right falls backward
Turning head to left falls forward
Attempt to overthrow normal pelvic girdle reactions

### TURNING

To RIGHT →
Amp. Fair
Duration 16 Sec

To RIGHT
Shoulder from above 8" to Right 6" to Right

Nystagmus 2/3 of normal
Vertigo 2/3 of normal (16 seconds.)
Past-pointing 2/3 of normal

To LEFT ←
Amp. Fair
Duration 8 Sec

To LEFT
Shoulder from above 3" to Left 4" to Left.

Nystagmus 1/3 of normal
Vertigo 1/3 of normal (8 seconds.)
Past-pointing 1/3 of normal

### CALORIC

Douche RIGHT None
Amp.
After 5 min. sec.

Douche RIGHT
Shoulder from above to Touched to Touched

Nystagmus None
Vertigo None
Past-pointing None
Falling No increase of spontaneous falling to Right

Head Back No nystagmus, no vertigo to Touched to Touched
Amp.

Douche LEFT None
Amp.
After 5 min. sec.

Douche LEFT
Shoulder from above to Touched to Touched

Nystagmus None
Vertigo None
Past-pointing None
Falling No change of spontaneous falling to Right

Head Back ← 8" to Left 10" to Left
Amp. Good

Head forward → 10" to Right 8" to Right.

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# CHAPTER XXIII

## PATHOLOGIC CASES ANALYZED

By Lewis Fisher, M.D.

It is presumed that the reader has made himself thoroughly familiar with the proper technic of the vestibular tests and with the significance of the various findings obtained as presented in the previous chapters. With the data properly recorded on a chart so that all the salient features of the examination can be taken in at a glance, how are we to proceed with the actual analysis of a case? The first problem in any given case is whether we are dealing with a functional or an organic condition. If the chart shows all responses to ear-stimulation perfectly normal, a functional condition may be suspected. When the responses obtained on stimulation are not normal, the case should be considered as having an organic lesion. Such a deviation from the normal need not include all the responses. An impairment of even one response shows that we are probably dealing with an organic lesion.

Having concluded that the case presents an actual organic involvement of the vestibular apparatus, our next problem is to determine whether the case is one of peripheral or central lesion. This is the most important and at the same time most difficult differentiation that the otologist is called upon to make. Many cases of cerebellar lesion or tumors of the cerebello-pontile angle present symptoms similar to those observed in an affection of the labyrinth—the miscalled "Menière's disease"; on the other hand, labyrinthine lesions not infrequently simulate cerebellar affections. The findings obtained upon ear stimulation are frequently the deciding factor in the diagnosis, and it therefore behooves the otologist to exercise the greatest care in determining this point. This subject is discussed in great detail in Chapters VI and XXI, but it might not be amiss to emphasize a few of the most important principles in this differential diagnosis.

In a peripheral lesion *all* the responses are impaired, and conversely the presence of any one *normal* response to stimulation suggests a normal labyrinth and VIII nerve. A spontaneous nystagmus in the vertical plane, either upward or downward, indicates a central lesion. If stimulation produces a "perverted" nystagmus then again the lesion is probably central.

If the findings lead to the conclusion that the lesion is central, then our next problem is to attempt to locate the lesion more definitely within the cranium. The facility with which one is able to do this depends in a great measure upon how well he can visualize the various pathways constituting the vestibular apparatus. The simplest method of procedure is that of elimination. We begin with the labyrinth and proceed brainward, considering each structure by itself. With good hearing and one or more responses on stimulation of the kinetic-static portion of the labyrinth normal, the labyrinth itself and VIII nerve may be considered uninvolved. For information relative to the medulla oblongata and inferior cerebellar peduncles, we examine the responses obtained on stimulation of each horizontal canal separately. This test is performed routinely by tilting the head back 60° after douching. If this produces normal horizontal nystagmus and vertigo with past-pointing, these structures are probably not involved. To determine the integrity of the pons, we examine the responses obtained from the vertical semicircular canals. These are tested when the ear is douched with the head in the so-called "upright" position. If the chart shows a normal rotary nystagmus with vertigo, past-pointing and falling, it suggests uninvolved pathways in the pons and middle cerebellar peduncle of the side douched. The cerebellum may be considered as not the seat of any gross lesion, if stimulation of either ear or any canal produces a past-pointing of both arms in both directions.

With the chart critically examined in this manner and all the possible points of involvement along the nerve tracts in mind, we attempt to find one location the involvement of which would satisfactorily account for all of the impaired responses. Thus, when the stimulation of all the semicircular canals of both sides produces no vertigo whatever or impaired vertigo, instead of assuming the presence of a great many lesions, it is more reasonable to suppose that there is one lesion at a point where all the fibres concerned in vertigo come together. Such a point is found at the decussation of the superior cerebellar peduncles where all of the fibres concerned pass from the cerebellum on their way to the cerebral cortex. Again, given a case with no responses at all from stimulation of the right ear, and an absence of responses from the vertical canals of the opposite or left ear, instead of assuming two lesions—one located in the right labyrinth, cutting off all the responses from that side, and another lesion in the left side of the pons—it is more reasonable to explain the whole phenomenon-complex by a single lesion in the right cerebello-pontile angle, where an involvement of the right VIII Nerve within this lesion would block all responses from the right labyrinth, and the same lesion by pressure against the brain-stem would interfere with the responses from the vertical semicircular canals of the opposite side.

Just as in Neurology a certain group of symptoms occurring with a definite lesion are spoken of as the "symptom-complex" for that lesion—in the same way a constant group of phenomena always appearing on ear stimulation in a certain lesion may be regarded as the "phenomenon-complex" for that particular lesion. The following should be remembered:

(1) If the results of stimulation of the right ear are: Nystagmus, none; vertigo, none; past-pointing, none; falling, none, it obviously indicates a destruction of the right labyrinth or VIII Nerve (see Fig. 97). We would of course have the corroborative evidence of complete deafness in this ear. This picture of a dead auditory apparatus and a failure of any of the semicircular canals to produce responses, shows beyond all doubt that the labyrinth or VIII Nerve is destroyed.

(2) If stimulation of the right horizontal canal produces: Nystagmus, none; vertigo, normal; past-pointing, normal; falling, normal; it suggests a lesion in the medulla oblongata between Deiters' nucleus and the posterior longitudinal bundle on the right side (see Fig. 98).

(3) If stimulation of the right horizontal canal produces: Nystagmus, normal; vertigo, none; past-pointing, none; falling, none; it suggests a lesion of the right inferior cerebellar peduncle (see Fig. 99).

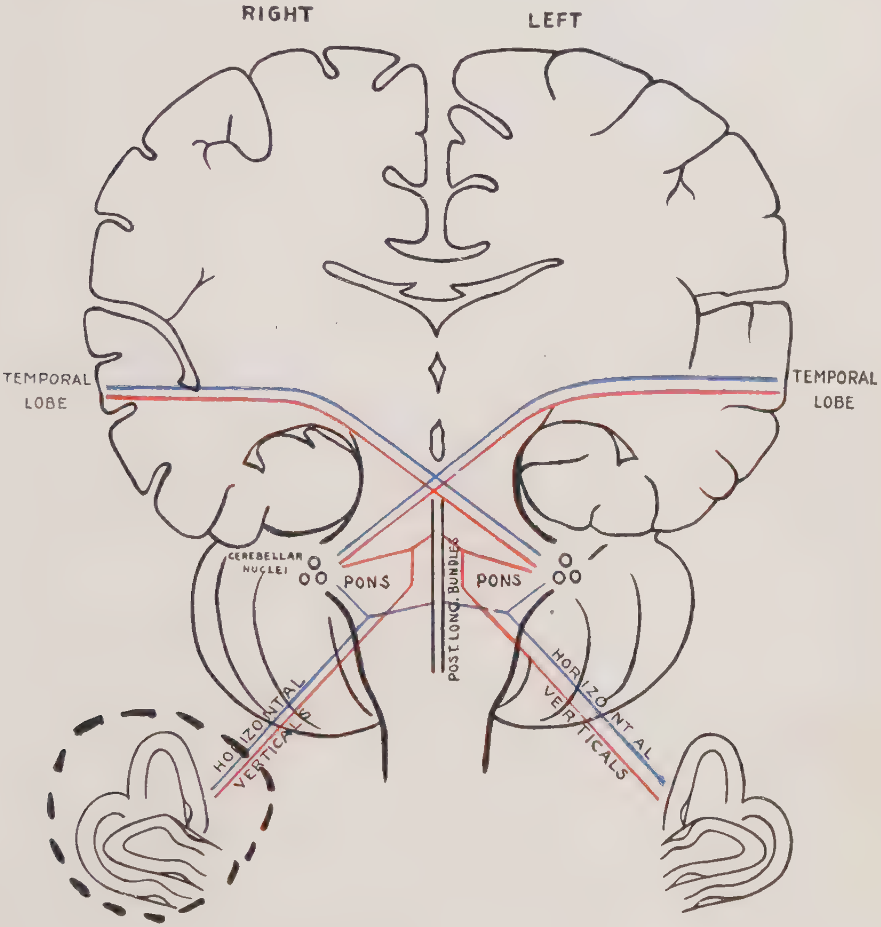

FIG. 97.—Dotted lines indicate lesion.

(4) If stimulation of the right vertical canals produces: Nystagmus, none; vertigo, normal; past-pointing, normal; falling, normal; it suggests a lesion in the posterior portion of the pons near the posterior longitudinal bundle on the right side (see Fig. 100).

(5) If stimulation of the right vertical canals produces: nystag-

mus, normal; vertigo, none; past-pointing, none; falling, none; it suggests a lesion of the right middle cerebellar peduncle (see Fig. 101).

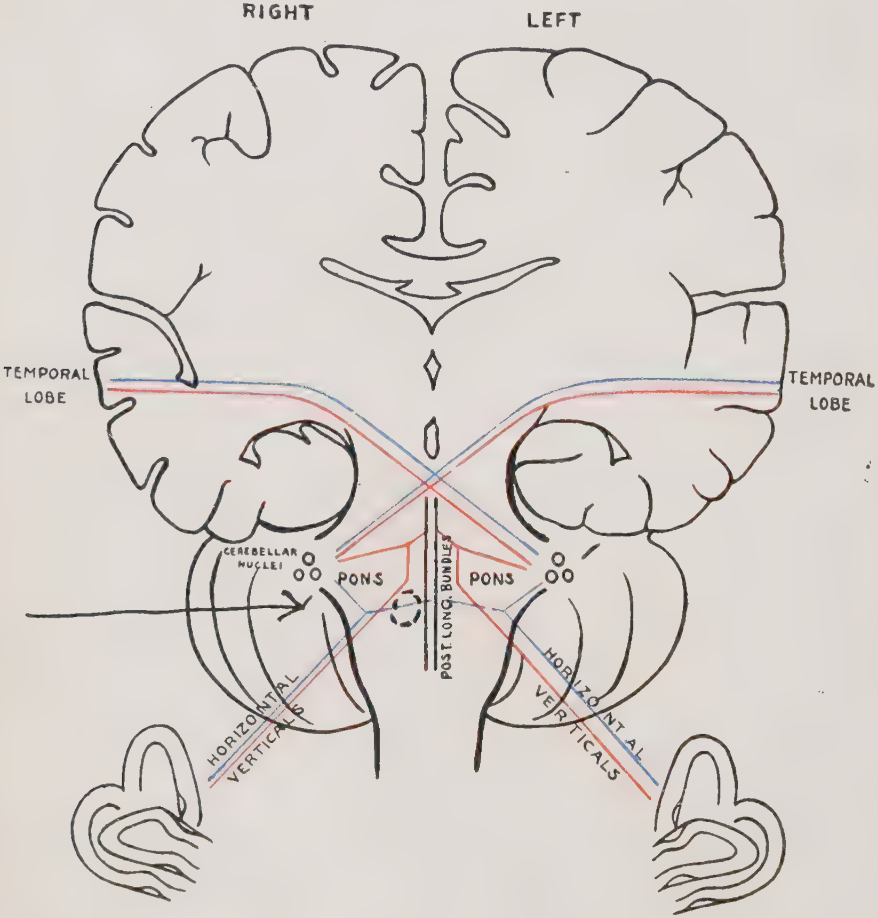

FIG. 98.—Arrow points to lesion.

(6) If stimulation of the horizontal canals of both ears and also of the vertical canals of both ears produces: Nystagmus, none; vertigo, normal; past-pointing, normal; falling, normal; it suggests a lesion of the posterior longitudinal bundle itself (see Fig. 102).

(7) If stimulation of the right horizontal canal and also of the right vertical canals produces: Nystagmus, normal; vertigo,

none; past-pointing, none; falling, none; it suggests a lesion of the cerebellar vestibular nuclei of the right side (see Fig. 103).

(8) If stimulation of all the semicircular canals of both ears produces: Nystagmus, normal; vertigo, none; past-pointing, none; falling, none; it suggests a lesion at the base of the cerebral crura at the point of decussation of the two superior cerebellar peduncles (see Fig. 104).

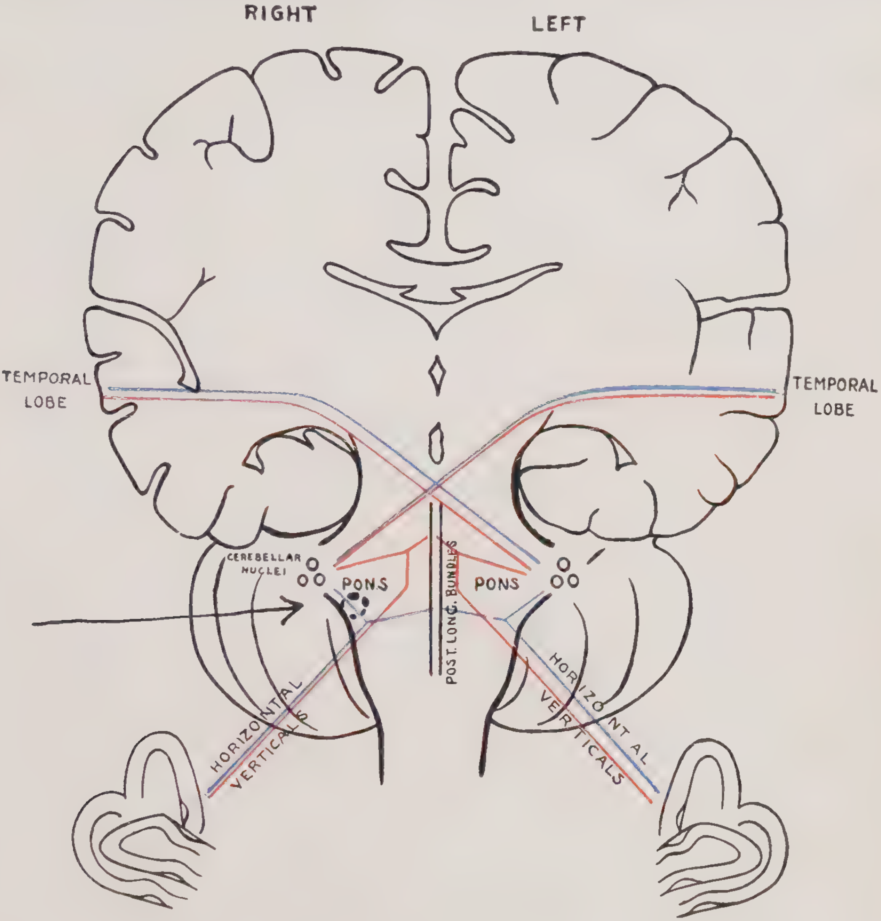

Fig. 99.—Arrow points to lesion.

(9) If there is total deafness in the right ear and stimulation of the right horizontal semicircular canal and of the right and left

vertical semicircular canals produces: Nystagmus, none; vertigo, none; past-pointing, none; falling, none; and stimulation of the *left* horizontal semicircular canal produces normal reactions, the lesion is located in the right cerebello-pontile angle (see Fig. 105).

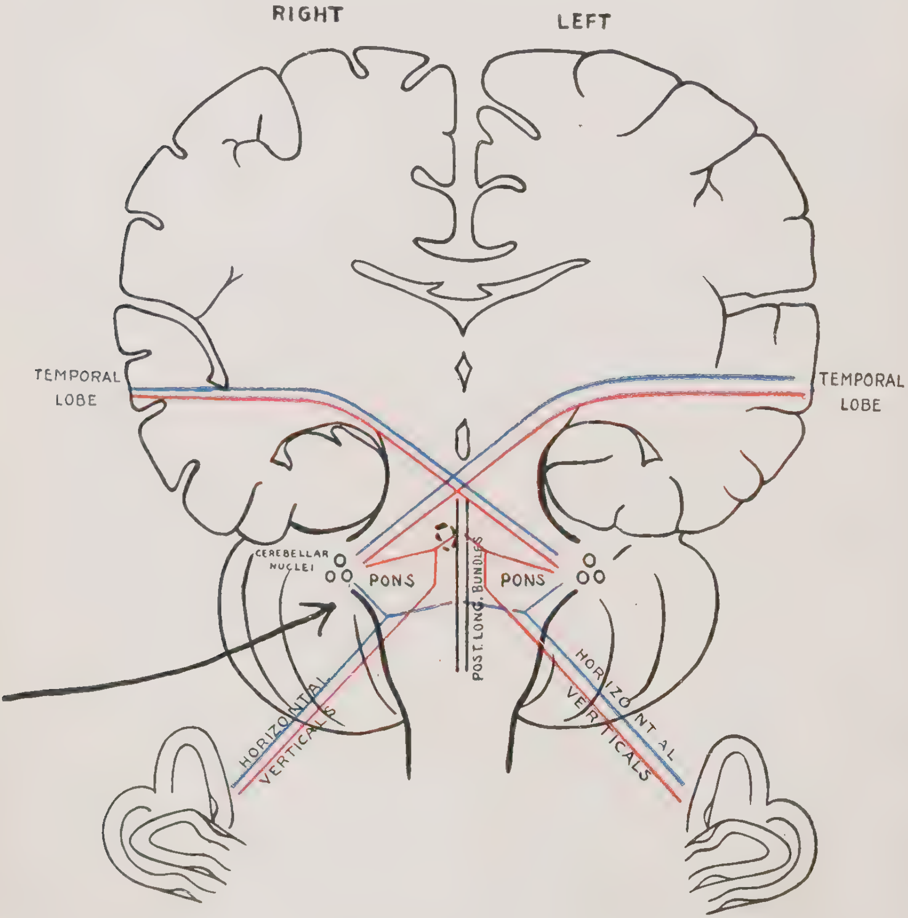

Fig. 100.—Arrow points to lesion.

It will be understood, of course, that actual lesions do not always fit neatly into the above schemata. As in medical diagnosis generally, all symptoms must be considered in their relation to other symptoms and that lesion postulated which will best explain the completed picture, just so in aural examinations relative values must be given to the various findings before a conclusion is reached. A lesion, for example, may involve more than one of the typical locations given in the charts, say the cerebello-pontile angle and the cerebellum itself; furthermore, the interference with the normal reactions may be due to direct destruction, or to transmitted pressure or both. Hence diagnoses cannot be made by rule of thumb, but must be based on a practical analysis of all the findings.

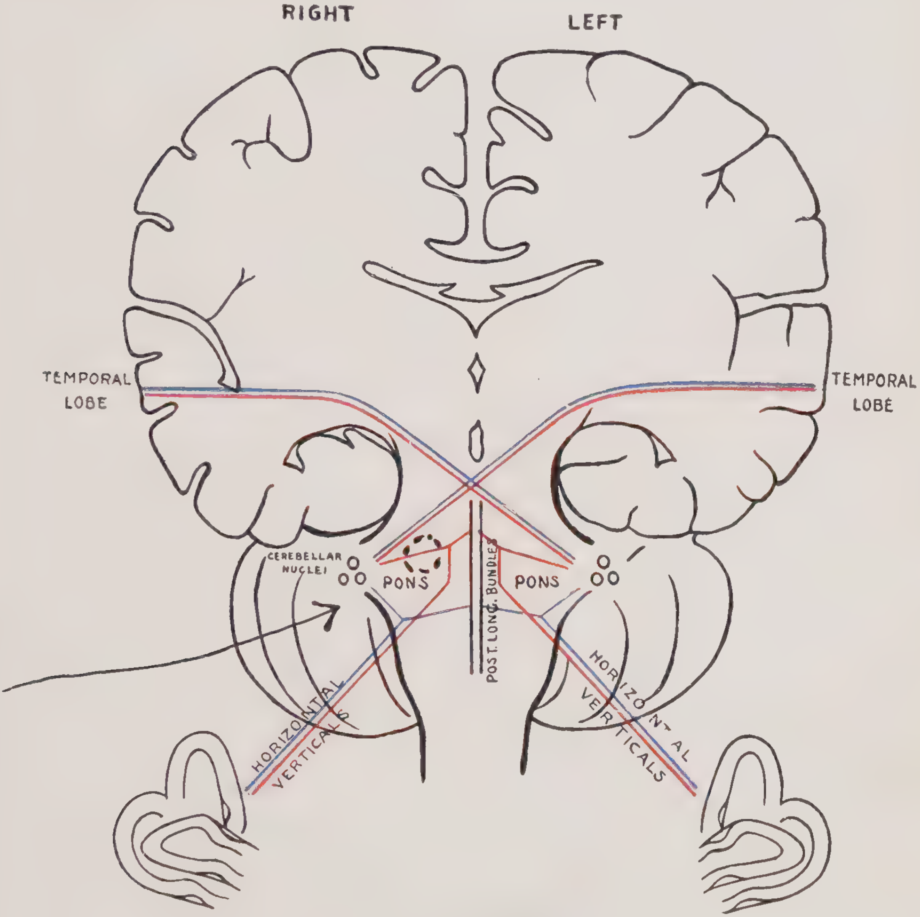

FIG. 101.—Arrow points to lesion.

In order to make clear the method of analysis, the following cases have been classified according to the site of the lesion, beginning with the middle ear, continuing to the internal ear, the VIII Nerve, the medulla oblongata, the pons, cerebellum, cerebrum and cerebello-pontile angle. The general broad division consists of peripheral and central lesions. The peripheral lesions include those of the labyrinth and the VIII Nerve while the central lesions include the remainder of the cases.

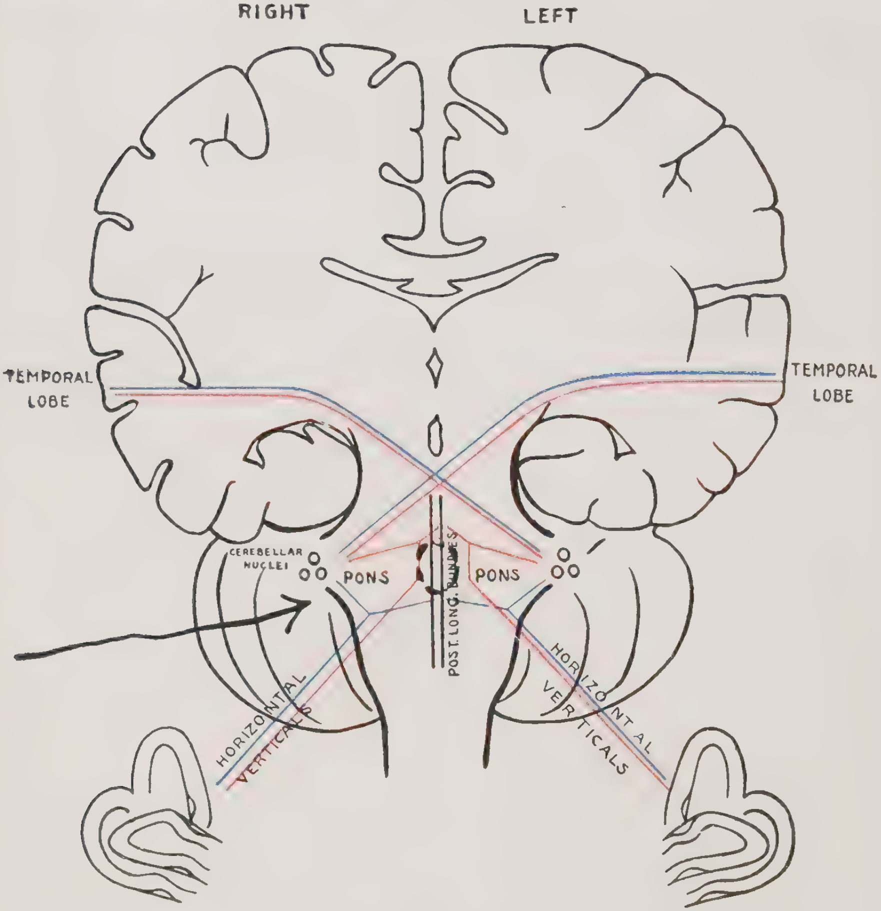

Fig. 102.—Arrow points to lesion.

Case 1.—Mr. John B., age 50. This case, so far as we have been able to ascertain, is the first demonstration in the living human being of the experiment which Ewald originally performed on pigeons.

The following is the patient's history as given in his own words:

"As far back as I can remember, I have had a running ear. My parents told me that it originally came from an attack of scarlet fever when a child. The discharge was not constant. It would appear occasionally, lasting for three or four months, and then would disappear for a period of four or five years. In June, 1916, the ear began to have this discharge, the only difference being that a little white kernel came out. This looked like a little mass of maggots without life. I merely noticed this little mass, but I suffered no inconvenience, and thus matters stood until the beginning of August, 1916. I then felt a slight irritation in the outer part of my right ear. I placed my finger on the outside and rubbed gently to alleviate this irritation. To my intense surprise, the whole room—everything in sight, in fact—revolved rapidly from the left to the right, and as quickly

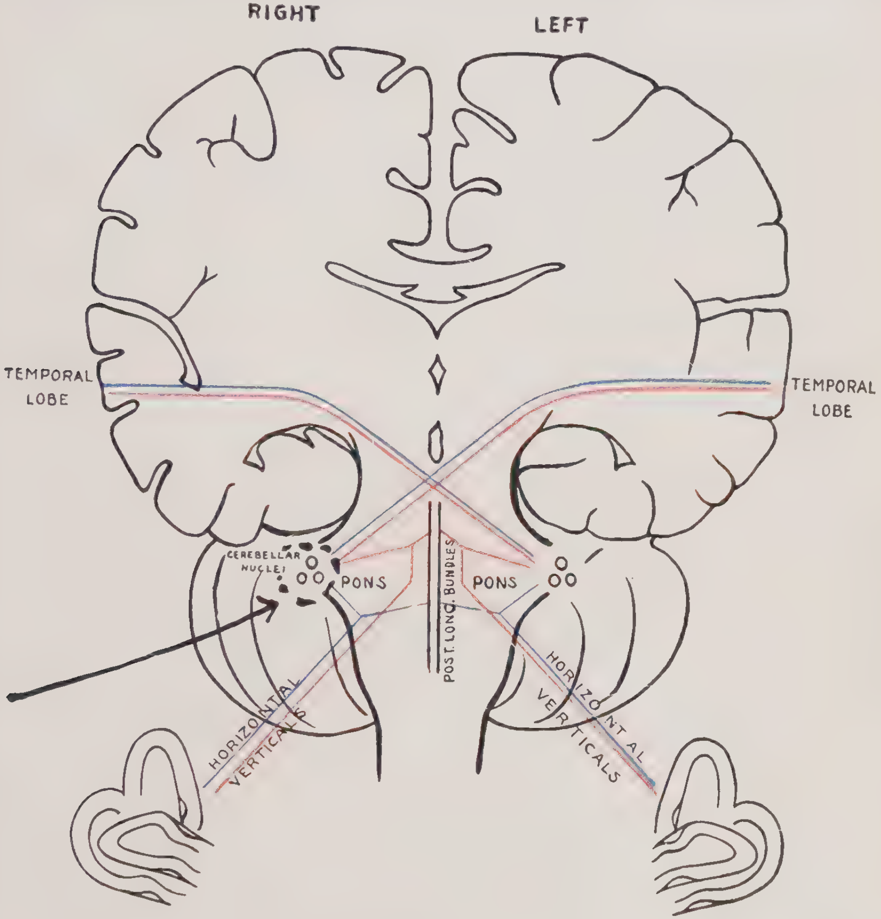

FIG. 103.—Arrow points to lesion.

back again. Thinking that this was only a delusion, I tried this 'push-button method' several times, and not once did it fail to register what I have just stated. I am a railroad conductor, and I have tried this same experiment while on the train. While standing on the rear platform of one of our cars I rubbed my ear and the entire train seemed to leave the track, swing over to the right, and swing back again quickly. This swinging of the train, first to the right and then back again, seemed to occur with myself acting as the axis of the turning. However, I paid no more attention to this matter, merely making it a point to keep my finger away from my ear, hoping that this queer condition might gradually disappear. A few mornings later, however, I was lying in bed on my back, and as I rolled over on my right side, it seemed as though some giant had lifted the left side of the

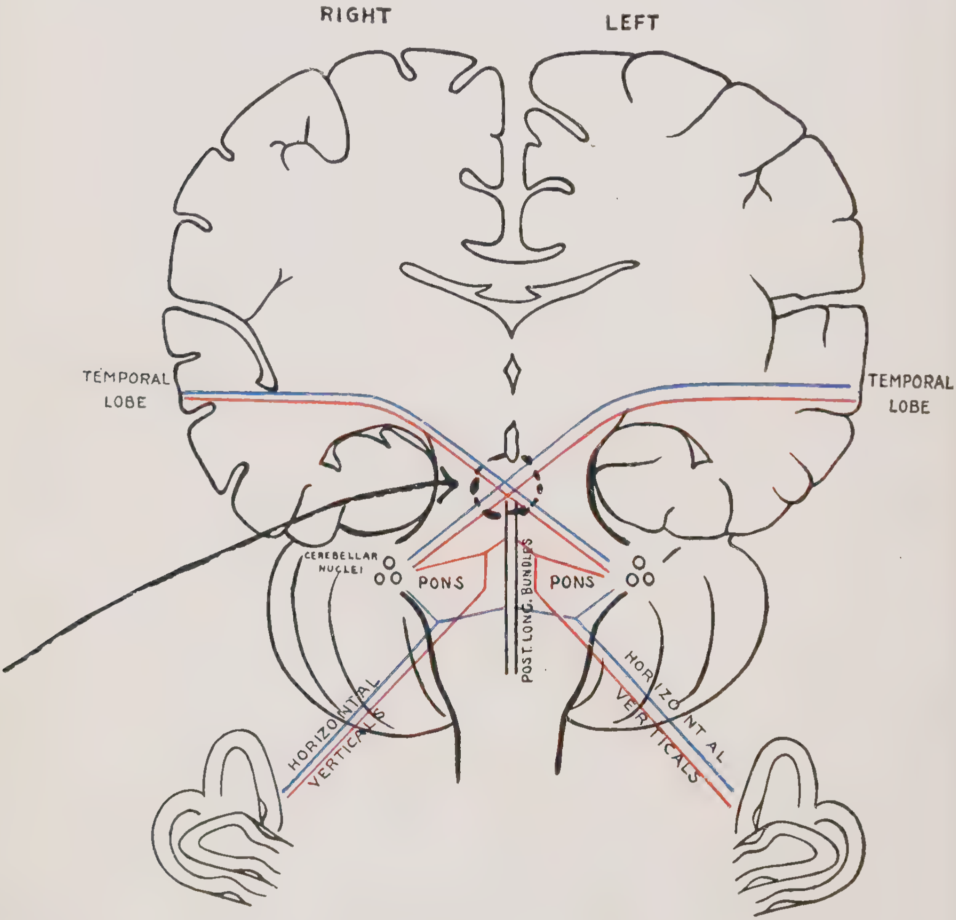

Fig. 104.—Arrow points to lesion.

bed to turn it over to the right, on top of me. I made a grab for the bed-covers. By this time I had stopped turning over to the right, and the elevating of the bed stopped at the same time. I must confess that I now began to be a little bit scared, as I could not understand why these things should be. Several days later, while on duty, I was addressed by a passenger, and turned quickly to the right to answer his question, when, to my surprise, I found myself falling against the seats on the left side. This unsettled condition became more and more intense each day, until it got so bad that I could not even shake my head from side to side, in saying 'no' in conversation, without having this 'falling feeling' come over me. I now became thoroughly frightened, as I thought these symptoms must surely be the forewarning of paralysis. I therefore consulted our family

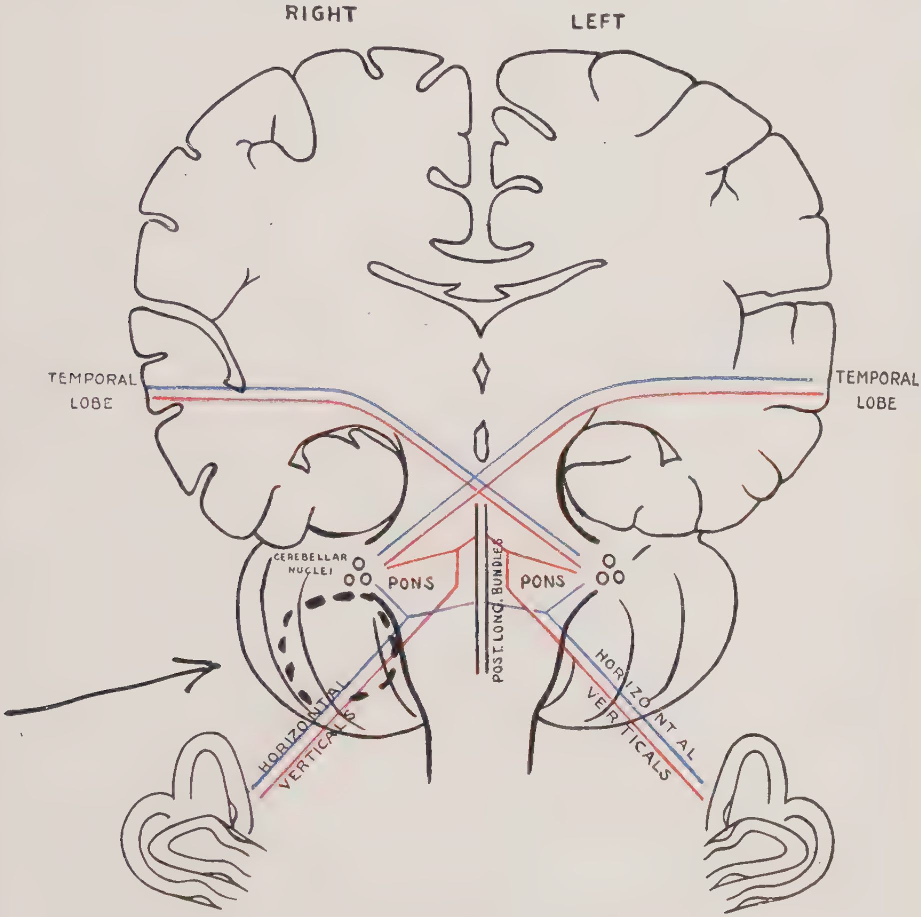

Fig. 105.—Arrow points to lesion.

physician, Dr. Victor Janvier, who told me that evidently there was something serious the matter with my ear."

*Ear Examination.* Right ear showed exacerbation of a chronic purulent otitis media, with a large perforation in the upper posterior quadrant. No polypi or granulation tissue. The pus was thick, creamy and fetid. There was necrosis of the upper posterior portion of the bony tympanic ring. The left ear showed evidence of a healed chronic purulent otitis media. Both cochleas were normal.

There was no spontaneous nystagmus or past-pointing. Turning to the right and to the left produced distinctly subnormal nystagmus, vertigo and past-pointing.

Pressure by a Politzer bag applied to the right auditory meatus caused a pure horizontal nystagmus to the right and a *systematized vertigo* also exclusively to the right. This could have been produced only by an endolymph movement exclusively to the left. The eyes were drawn to the left in the direction of the endolymph movement, and the quick, cerebral jerk to the right completed the nystagmus to the right. As the vertigo is always in a direction opposite to the endolymph movement, this endolymph movement exclusively to the left produced a systematized vertigo to the right. Because of this systematized vertigo, all of the extremities *past-pointed* to the left during the time that the pressure was applied to the ear.

The accompanying series of photographs (Figs. 106 to 110) shows the patient's ability to find the finger spontaneously. When the Politzer bag, however, is compressed, the patient is unable to find the finger, but "past-points" to the left with all extremities.

### COMMENT

It is a matter of common experience that in a certain number of cases of chronic purulent otitis media there is an extension of the process into the labyrinth, which is made evident by the appearance of a nystagmus when pressure is applied to the external auditory meatus. When a nystagmus appears on pressure, the fistula test is considered positive and usually indicates a fistulous opening into the labyrinth. The production of a nystagmus, however, proves merely that there is present an opening into the labyrinth at some point. In the original Ewald experiment an artificial plug was inserted into a semicircular canal and then a hole was drilled at a point adjacent to this plug. In this way Ewald was enabled to cause an endolymph current *in one direction only.* This patient has not only an opening into his horizontal semicircular canal, located at the point where the canal bends out-

FIG 106.

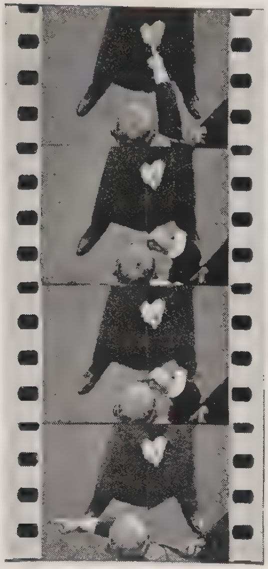

FIG. 107

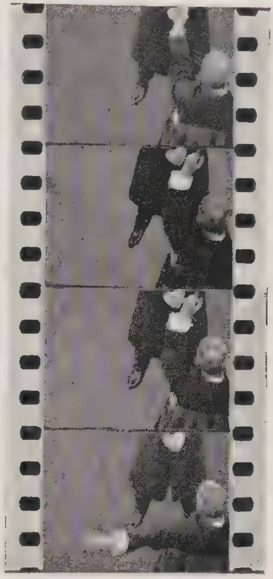

FIG. 106.—Pointing; right shoulder from above.

FIG. 107.—Pointing; left shoulder from above.

FIG. 108.

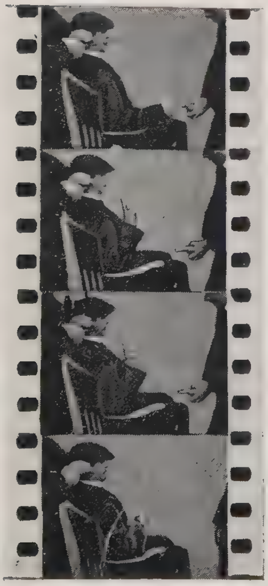

FIG. 109.

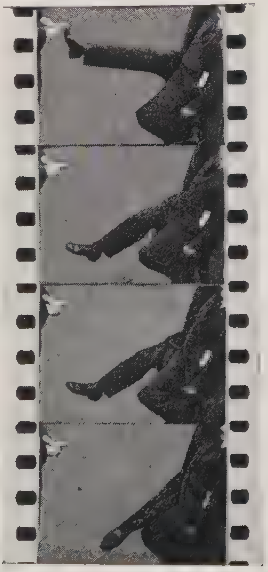

FIG. 110.

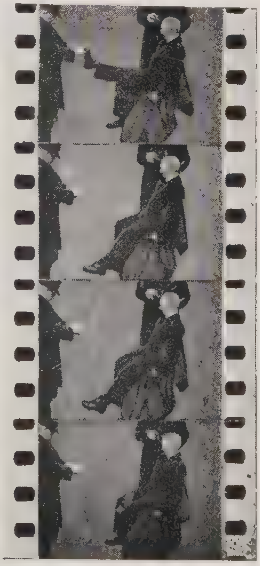

FIG. 108.—Pointing; elbow from above.

FIG. 109.—Pointing; right hip from below.

FIG. 110.—Pointing; left hip from below.

ward to produce the aditus ad antrum, but he also has a closure of the canal at a point behind the fistulous opening. This is demonstrated by the fact that pressure by the Politzer bag produces not only a nystagmus but a *systematized vertigo.* This systematized vertigo is not merely made probable by the intelligent statement of the patient himself that he *feels* that he is rotating to the right at a speed of forty miles an hour, but is also definitely proven in that he past-points to the left invariably, with all extremities, because of this vertigo.

Under local treatment the purulent discharge has disappeared, the symptoms have subsided and the fistula test is no longer positive.

Case 2.—Mrs. A. G., age 44. Patient was admitted to a Philadelphia hospital October 20, 1914, with the following history: Was in good health until three months before admission. The first thing noted was difficulty in walking; this continued for one month, when patient began to notice "trouble with the eyes." First she noticed occasional flashes of light before the eyes and in the course of two weeks more she realized that her vision was getting dim. One month before admission to the hospital she began to have attacks of vertigo, nausea and projectile vomiting, associated with severe occipital headache. The difficulty in walking has steadily increased, with a tendency to falling—always to the right side. She had never had a convulsion.

The record of the physical examination is as follows: "No tremor of extremities but slight ataxia of both arms and legs and slight hypermetry of the right upper extremity. Romberg—falls to the right. Can not stand on either foot; falls to the right. Delayed sensation, particularly on the right side of the body, and numbness on the dorsum of the right hand. The right pupil is slightly larger than the left and there is a slight drooping of the left eye-lid."

The patient had been sent to the hospital with a provisional diagnosis of brain tumor; this diagnosis was confirmed by the internists at the hospital and two neurologic consultants agreed that the history, symptoms and examination indicated a tumor of the right cerebellar hemisphere. This diagnosis was further confirmed by the X-ray report, which stated that the photographs showed a tumor in the right cerebellar hemisphere and that it appeared to be a cyst. All agreed that operation was indicated.

It was at this time that the ear examination was made. It is of peculiar interest to note that the reason an ear examination was suggested was not because any particular help was expected in making the diagnosis, but because of the unusual opportunity to determine what the ear tests would show in a case in which the diagnosis was already so clear and definite.

*Ear Examination.* Referring to the chart (XV), we note particularly that the *past-pointing* of both arms in both directions was not only present and in the correct direction, but that the past-pointing was even exaggerated in its extent. The lower extremities also past-pointed correctly in both directions. This exaggerated past-pointing in the correct directions was observed both after turning and douching.

Because of the newness of this type of examination, the following report was made with considerable misgiving: "The ear-tests, *per se,* would suggest that the cerebellum is normal. The pointing tests of the upper and lower extremities after stimulation of the labyrinths would seem to indicate normal cerebellar hemispheres; the tests for the neck and trunk also show normal responses, which suggests that the vermis is normal."

Because of the ear report, operation was postponed to await developments. The patient soon began to get better and better, was discharged from the hospital within a month, has since given birth to a healthy child and has remained in good health—at least up to the last time she was seen, which was nearly two years after her illness. As she has moved to another city, we do not know her present condition.

Because of the happy outcome in this case, the actual nature of the lesion is not determined. However, the case serves to illustrate how the ear tests may be of value in preventing unnecessary operations.

Case 3.—Mrs. W. B. W., age 51. Chief complaint, attacks of vertigo and staggering, impaired hearing and slight tinnitus.

Referred on February 4, 1916, with the following history: First attack of dizziness in 1897, lasting three minutes; she had three or four attacks shortly after that—approximately one every month. One attack in 1911 was quite severe, with considerable nausea, and lasted 24 hours. Two months later another attack occurred while on the street, but she was able to get home and the symptoms disappeared after three hours. In June, 1913, a slight attack, lasting ten minutes. In August, 1913, a more severe attack with nausea lasting 48 hours. In August, 1914, an attack of 48 hours' duration, with continued vertigo and nausea, and for one week patient was unable to retain anything in her stomach—not even a glass of water. For the next six months there were repeated attacks every month or so, each lasting four or five hours, usually with nausea.

In December, 1914, because of nausea, vomiting, emaciation and jaundice, an operation on the *gall bladder* was performed, malignancy having been suspected. The gall bladder was found to be normal. The attacks continued with more or less frequency--one attack occurring three weeks after the operation. November, 1915, while sitting in the theatre, patient was seized with a severe attack of dizziness, nausea and vomiting, and had to be taken from the theatre to the hospital, where she was confined to bed for seven days.

First noticed impairment of hearing in 1903. In 1911 deafness in the left ear became more noticeable and after the severe attack in November, 1915, she noticed that she was entirely deaf in the left ear. Patient had complained of noises in the head occasionally during the past ten years. After the attack in 1915 the noises have been louder; they are not continuous, however, and are only in the left ear.

## CHART XV A

Name Mrs. A. G. Age 44 Date Nov. 6, 1914.
Address
Referred by

DIAGNOSIS:

SUMMARY:

Complains of Vertigo, nausea and vomiting

HISTORY:
Dizziness Yes
Staggering Yes
Deafness No
Tinnitus No

NOSE: Chronic sinusitis

THROAT: Tonsils small, not pathologic. Teeth absent

EARS:
A. D. Almost immovable hammer-handle.
A. S. Impaired mobility of hammer handle

Fistula Negative

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | 3 seconds | | | | | | | |
| $\underline{\underline{A}}$ | ½ | A | 5 | Ac > Bc ≪ n | Pol. | | $c^4$ | Good | Galt | ·9 | Weber |
| | ½ | | 5 | Ac > Bc ≪ n | | | | Good | | :9 | — |
| | | | | 4 seconds | | | | | | | |

## CHART XV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT ↷ marked
Looking to LEFT ↶ marked
Looking UP None
Looking DOWN None
Straight ahead, none

|  | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 3" to Right | 1" to Left |

Nystagmus in extreme lateral positions
Vertigo Yes
Past-pointing Yes
Falling Yes to Right, with eyes open
Romberg falls to Right
Turning head to right not tested
Turning head to left not tested
Attempt to overthrow pelvic movements normal

### TURNING

To RIGHT →
Amp. Good
Duration 40 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 12" to Right | 12" to Right. |

Nystagmus exaggerated
Vertigo Exaggerated
Past-pointing exaggerated

|  | RIGHT | LEFT |
|---|---|---|
| Hip from below | 4" to Right | 5" to Right |

To LEFT ←
Amp. Good
Duration 38 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 12" to Left | 9" to Left |

Nystagmus Exaggerated
Vertigo exaggerated
Past-pointing exaggerated

|  | RIGHT | LEFT |
|---|---|---|
| Hip from below | 4" to Left | 4" to Left |

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 55 sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 12" to Right | 4" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Exaggerated
Falling Normal

| Head Back not tested | ~~to~~ | ~~to~~ |
|---|---|---|
| Amp. |  |  |

Douche LEFT ↶
Amp. Fair
After 1 min. 55 sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 12" to Left | 5" to Left. |

Nystagmus
Vertigo
Past-pointing
Falling

| Head Back not tested | ~~to~~ | ~~to~~ |
|---|---|---|
| Amp. |  |  |

## CHART XVI A

Name Mrs. H. B. H. Age 52 Date Feb. 4, 1916

Address

Referred by Dr Robert Pitfield.

DIAGNOSIS:

SUMMARY:

Complains of Attacks of vertigo for years.

HISTORY:

Dizziness Yes.

Staggering Yes

Deafness Yes, left ear

Tinnitus Yes, left ear

NOSE: Septal spur into left middle meatus

THROAT: Negative

EARS:

A. D. Impaired mobility of hammer-handle, cicatrix anterior inferior quadrant.

A. S. Impaired mobility of hammer-handle, cicatrix posterior to umbo

Fistula Negative

Hearing Tests

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3/4 | A | 7 | Ac | > | Bc | = | n | Pol. | — | $c^4$ | Good | Galt | 5. 3 | Weber |
| | 0 | | 0 | Ac | ? | Bc | < | n | | — | | None | | None | — |

## CHART XVI B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus
Vertigo
Past-pointing
Falling
Romberg
Turning head to right
Turning head to left
Attempt to overthrow

### TURNING

To RIGHT →
Amp. Fibrillary
Duration 8 Sec.

| To RIGHT | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 1" to Right | Touched |

Nystagmus almost none
Vertigo " "
Past-pointing " "

To LEFT ←
Amp. Better but poor
Duration 10 Sec.

| To LEFT | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 12" to Left<br>Touched | 4" to Left<br>Touched |

Nystagmus present but shortened
Vertigo " " "
Past-pointing " " "

### CALORIC

Douche RIGHT ↷
Amp. Small
After 2 min. 30 sec

| Douche RIGHT | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 5" to Right | 5" to Right. |

Nystagmus markedly impaired
Vertigo present
Past-pointing present
Falling subnormal but present

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Head Back →<br>Amp. Poor | 3" to Right | 4" to Right |

Douche LEFT ↶
Amp. Slight
After 2 min. 43 sec

| Douche LEFT | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 3" to Left | 3" to Left |

Nystagmus markedly impaired
Vertigo present
Past-pointing present
Falling subnormal but present

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Head Back ←<br>Amp. Fair | 3" to Left | 3" to Left |

*Ear Examination.*—Referring to the chart (XVI), we note impairment in nystagmus, vertigo, past-pointing and falling after turning and douching.

The following report was made:

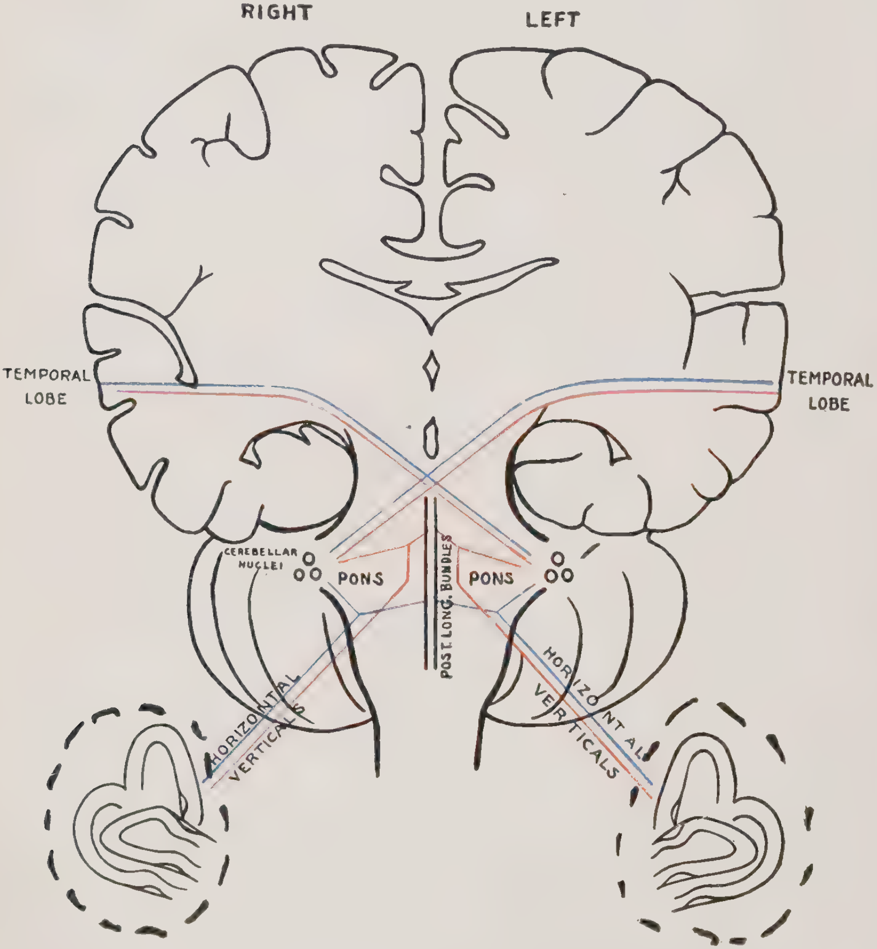

FIG. 111.—Dotted lines indicate location of the lesion.

Right ear: (1) Cochlea normal, for all practical purposes: there is, however, a distinct impairment for the very highest tones. This suggests that there has been a disturbance in the cochlea which has cleared up. (2) Static labyrinth. Distinct impairment of the horizontal and also the vertical semicircular canals.

Left ear: (1) Cochlea "stone dead." (2) Static labyrinth. Marked impairment of the vertical semicircular canals. Moderate impairment of the horizontal semicircular canal.

*Conclusions.*—The attacks have been caused by a toxic process in both *internal ears* (Fig. 111).

*Prognosis.*—"For ultimate recovery, prognosis is very good. She will, however, have other attacks unless the source of the toxæmia can be found. A mere congestion of ischæmia of the labyrinth would hardly account for the definite degeneration that has taken place in the labyrinths."

Her physician considers that the toxæmia which caused the labyrinthitis was of intestinal origin, and he noticed that whenever she ate eggs or fish she almost invariably had vestibular symptoms. Indicanurea was frequently demonstrable. Under dietetic treatment the patient has improved, and for the past year, to date, has been free from any attacks.

Case 4.—Mr. D. L. V., age 32. Chief complaint—tinnitus, impaired hearing, vertigo and staggering. Referred by Dr. Alfred Stengel with the following history:

While at the Plattsburg Training Camp on October 1, 1916, patient was somewhat stunned by the explosion of a rifle near his left ear. Thirteen days later he began to be dizzy. This vertigo was constant for two weeks, but after that was noticeable only when standing or walking. When he lay down he noticed no "swimming of the head," but a slight sensation of nausea, similar to that of seasickness. Staggering was quite marked for the first two weeks and was more noticeable in the dark. At the time of the attack, October 13th, patient also noticed sudden impairment of hearing and noises in the left ear, similar to the rapid ringing of a small shrill bell at a considerable distance.

*Ear Examination.*—Referring to the chart (XVII) we note that the hearing tests are suggestive of the very slightest nerve impairment of both ears. Patient has himself observed that the recent impairment of hearing in the left ear has largely disappeared. Turning and douching show markedly impaired nystagmus, vertigo, past-pointing and falling—in other words *all* responses from both ears are impaired.

The following report was made—"*Summary*—Impairment of both internal ears (Fig. 112), especially the left. In detail:

(1) Right ear. (*a*) Cochlea normal, except in its uppermost register, which is impaired. (*b*) Semicircular canals distinctly subnormal. The vertical canals are impaired 1/3 of normal function; horizontal canal shows similar impairment.

(2) Left ear. (*a*) Cochlea normal except for the very highest tones; these upper tones are somewhat more impaired than in the right cochlea. (*b*) Semicircular canals almost completely destroyed. The vertical canals show merely a trace of function and the horizontal canal is completely dead.

*Etiology.*—The definite history of noticeable freedom from seasickness all his life suggests that this impairment of his internal ears occurred when he was a child. It may have been caused by mumps. The recent attack on October 13th, causing deafness, tinnitus, vertigo and staggering, was due to additional involvement of the left ear only. The explosion of the rifle may have caused a concussion of the left internal ear, but it must be noted that the tinnitus and vertigo did not occur immediately after the explosion.

*Prognosis.*—(1) Hearing—good prognosis. The recent impairment of hearing in the left ear has cleared up very noticeably in the past two weeks; this shows that

## CHART XVII A

Name Mr. D. L. V Age 32 Date Nov. 11, 1916.
Address
Referred by Dr Alfred Stengel

DIAGNOSIS:

SUMMARY:

Complains of Vertigo. and of "Feeling uncertain of himself."

HISTORY:
Dizziness Yes
Staggering Yes
Deafness Slight, left ear
Tinnitus Slight, left ear

NOSE: Slight atrophic rhinitis

THROAT: High arched palate Teeth in poor condition

EARS:
A. D. Normal. Unusually perfect movement of ossicles
A. S. Slight retraction of membranous tympani otherwise normal

Fistula Not tested

Hearing Tests

| | | | | | | |
|---|---|---|---|---|---|---|
| $\underset{=}{A}$ | A | | Pol. | $c^4$ | Galt | |
| 2 | 7 | Ac > Bc = n | | Good | 1.8 | |
| 2½ | 7 | Ac = Bc = n | | Good | 2.4 | $\overline{\text{Weber}}$ ? |

## CHART XVII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling None
Romberg negative
Turning head to right negative
Turning head to left negative
Attempt to overthrow normal pelvic movements

### TURNING

To RIGHT →
Amp. Poor
Duration 2 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus practically none
Vertigo none whatever, not a "sensation".
Past-pointing absent

To LEFT ←
Amp. Poor
Duration 8 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 3" to Left | Touched |

Nystagmus almost none
Vertigo, subnormal
Past-pointing, just the least suggestion

### CALORIC

Douche RIGHT ↷
Amp. Fair
After 1 min. 23 sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 2" to Right | 1" to Right |

Nystagmus subnormal
Vertigo subnormal
Past-pointing subnormal
Falling subnormal

Head Back →
Amp. Fair

| | RIGHT | LEFT |
|---|---|---|
| Head Back | 1" to Right | 1" to Right |

Douche LEFT ↶
Amp. Barely perceptible
After 2 min. 56 sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | ½" to Left | Touched |

Nystagmus Almost none
Vertigo " "
Past-pointing " "
Falling Absent

Head Back
Amp. no nystagmus

| | RIGHT | LEFT |
|---|---|---|
| Head Back | Touched | Touched |

there was either a concussion or mild toxæmia of the cochlea. (2) Vertigo—prognosis also good. The cerebrum should very readily adapt itself to this ear impairment, and it is probable that within a month the patient should be free from all dizziness and inco-ordination.

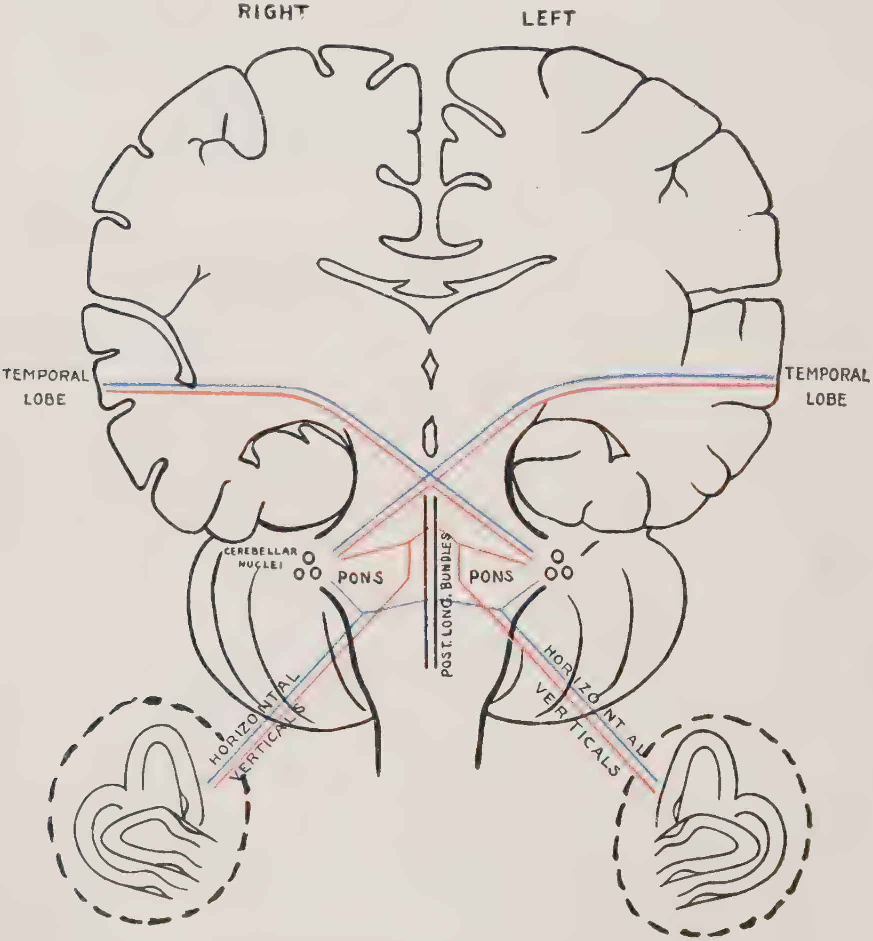

Fig. 112.—Dotted lines indicate location of lesion.

This opinion was justified by the outcome of the case—complete recovery from all symptoms.

Case 5.—Dr. C. W., age 53. Chief complaint, continual tinnitus. Referred by Dr. William Gerry Morgan with the following history:

Patient noticed noises in the head during the winter and spring of 1909. In the fall of 1909, while on board a vessel crossing from Korea to Japan, he was seized with what he thought to be seasickness. The vertigo, however, persisted for two weeks. It is to be noted also that the tinnitus was increased at the time of this attack. Vertigo and unsteadiness of gait disappeared after the two weeks, but the tinnitus has continued to the present date, March 30, 1917. In 1911, while in South America, for two months patient was constantly annoyed with a sensation that he wanted to go to the toilet, and yet there was very little actual diarrhœa. He attributed this to climatic change.

*Ear Examination.*—Referring to the accompanying chart (XVIII), we note that the tuning-fork tests show impaired hearing of the nerve type, and the nystagmus, vertigo, past-pointing and falling, after both turning and douching, are impaired. These findings may be summed up as follows:

(1) Right Ear. (*a*) Cochlea shows one half of normal function. (*b*) Vestibular labyrinth. (*x*) Horizontal canal slightly impaired. (*y*) Vertical canals slightly impaired.

(2) Left Ear. (*a*) Cochlea impaired one-third of normal function. (*b*) Vestibular labyrinth. (*x*) Horizontal canal slightly impaired a little less than the right horizontal canal. (*y*) Vertical canals very slightly impaired.

The following report was made: "The proportionate impairment in both ears of both the cochlear and vestibular portions indicates, first, that some general disturbance such as a toxæmia has affected both portions of both internal ears (Fig. 113), and, second, that the intracranial pathways from the ear are intact throughout their course through the brain-stem, cerebellum and cerebrum. The impairment of the vestibular portion of each internal ear is in no sense a practical drawback to his general well-being. The tests by turning, douching and the galvanic current all agree in that each type of stimulation produces subnormal responses in nystagmus, vertigo, past-pointing and falling. This impairment, however, is very slight and does not in any way affect his equilibrium. The cause of the original attack was most probably not seasickness, but a toxic labyrinthitis involving both internal ears. This internal ear disturbance had apparently begun in slight degree the previous winter and spring, and was evidenced by the tinnitus which began at that time. The peculiar sensations noticed in South America were probably not climatic, but due to the toxic irritation of the internal ears.

*Prognosis.*—Excellent for hearing, guarded for the cessation of the tinnitus. There is no reason to anticipate any increase in the impairment of hearing. We are not dealing with a progressive condition; the mischief was done nine years ago, once and for all. Similarly we need have no fear that the noises will increase in severity, and it may reassure the patient to tell him this. The prognosis, therefore, is very favorable, with the single exception that the slight tinnitus which he now observes when he is in a very quiet place or when he is physically fatigued will in all probability continue."

The essential point in this case is that the vestibular tests were helpful in rounding out the knowledge of the cause of the tinnitus by furnishing an additional method of approach in investigating the condition of the labyrinths.

Case 6.—Mr. James H., age 38. Referred by Dr. Charles H. Frazier on January 7, 1916, with the following history: On January 25th, 1913, patient was oper-

## CHART XVIII A

Name Dr. C. W. Age 53 Date March 30, 1917.
Address
Referred by Dr Wm Gerry Morgan.

DIAGNOSIS:

SUMMARY:

Complains of Tinnitus.

HISTORY:
Dizziness No
Staggering No
Deafness No
Tinnitus Yes

NOSE: Nares slightly atrophic posteriorly.

THROAT: Tonsils buried Slight amount mucoid material in crypts of right.

EARS:
A. D. Merely a slight impairment of mobility of hammer-handle
A. S. Same, and slight retraction of M. T.

Fistula Negative

Hearing Tests

distinct

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 1/4 | A | 4 | Ac > Bc < n | Pol. | — | $c^4$ | Good | Galt | .7 | Weber ± |
| | 1/2 | | 5 | Ac > Bc < n. | | — | | Good | | 1.7 | |

slight

## CHART XVIII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT *None*
Looking to LEFT *None*
Looking UP *None*
Looking DOWN *None*

Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| Touched | Touched |

Nystagmus *none*
Vertigo *none*
Past-pointing *none*
Falling *none*
Romberg *Negative*
Turning head to right *Negative*
Turning head to left *Negative*
Attempt to overthrow *Negative*

---

### TURNING

To RIGHT →
Amp. *Good to fair*
Duration *19* Sec.
*Unusually rapid*
*Somewhat irregular*

To RIGHT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| 14" to Right | 6" to Right |
| Touched | 1/2 to Right |
| Touched | Touched |

Nystagmus *Impaired*
Vertigo *Impaired – 13 seconds*
Past-pointing *Impaired*

To LEFT
Amp. *Good to fair*
Duration *25* Sec.
*Rapid and irregular*

To LEFT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| 20" to Left | 6" to Left |
| 2" to Left | 2" to Left |
| Touched | Touched |

Nystagmus *Slightly impaired*
Vertigo *Slightly impaired – 19 seconds*
Past-pointing *Slightly impaired*

---

### CALORIC

Douche RIGHT ↷
Amp. *Good*
After *1* min. *40* sec.

Douche RIGHT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| 4" to Right | 4" to Right |

Nystagmus *Delayed*
Vertigo *Slightly subnormal*
Past-pointing *Somewhat subnormal*
Falling *Slightly subnormal*

Head Back →
Amp. *Good*

| RIGHT | LEFT |
|---|---|
| 5" to Right | 4" to Right |

Douche LEFT ↶
Amp. *Good*
*3/17/17* After *1* min *10* sec.

Douche LEFT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| 4" to Left | 5" to Left |

Nystagmus *Slightly delayed*
Vertigo " "
Past-pointing *Slightly subnormal*
Falling *Subnormal*

Head Back ←
Amp. *Good*

| RIGHT | LEFT |
|---|---|
| 4" to Left | 6" to Left |

ated on at the hospital of the University of Pennsylvania for persistent tinnitus following a fracture at the base of the skull. Doctor Frazier divided the left VIII Nerve in the posterior fossa. Shortly after the operation all the roaring and ringing almost entirely disappeared. After the patient had enjoyed freedom from symptoms for four months, however, the tinnitus returned.

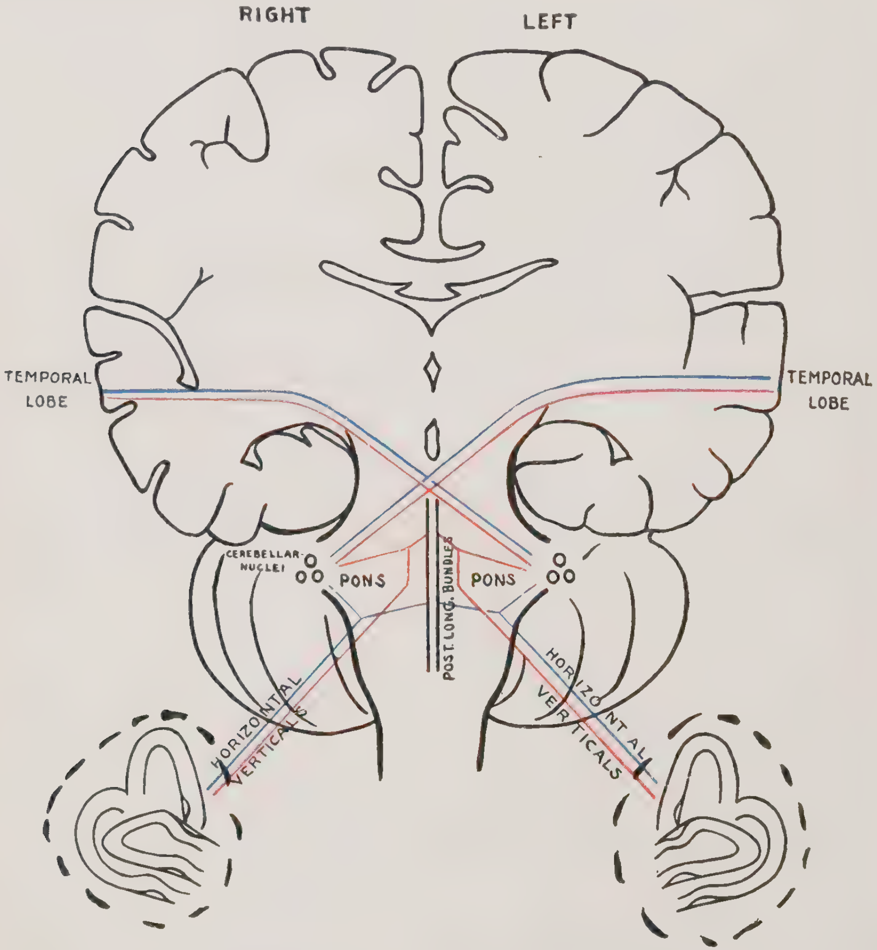

FIG. 113.—Dotted lines indicate location of lesion.

Referring to the chart (XIX), we note that this ear-examination was made three years after the operation. The findings indicate a completely non-functionating left ear, for the following reasons:

(1) The tuning-fork tests show complete deafness in the left ear.

(2) Douching the left ear fails to produce any of the responses in nystagmus, vertigo, past-pointing and falling.

(3) Turning to the right, which stimulates chiefly the left ear, causes a very poor nystagmus, vertigo and past-pointing, whereas turning to the left, which stimulates particularly the right ear, shows fairly good responses.

This examination indicates that the operation not only completely severed the VIII Nerve, but there has been no regeneration either of its cochlear or vestibular portions. This case is of interest in that it may throw light upon some phases of the subject of tinnitus. At first glance it would seem to bear out the contention of those who maintain that tinnitus is caused by a lesion within the labyrinth. For four months after the severing of the VIII Nerve the patient was comparatively free from noises in the head. The recurrence of the tinnitus, however, shows without question that in this case at least there occurred after the severing of the VIII Nerve a further degenerative process, *central* to the point of section, and capable of producing tinnitus. Before operation it would seem reasonable to consider that the process attacked the cells within the spiral ganglia peripheral to the point of section. This would account for the patient's temporary recovery from the tinnitus. The ear-tests now show that the VIII Nerve was completely severed, and yet the tinnitus recurred. This would indicate that the tinnitus is most probably produced by a further toxic involvement of the ganglionic cells along the course of the auditory nerve-tracts or at their termini in the cerebral cortex.

CASE 7.—Charles W. C., age 21, gave the following history: About two years ago while a sailor on board a United States battleship, he fell from the upper bridge, a distance of approximately 40 feet, first striking the back of his head and then the right side. The fall rendered him unconscious for 6 days and 8 hours and produced severe contusions of his mouth, nose and ears. When he recovered consciousness he found that he was absolutely deaf. When he felt strong enough to get out of bed he discovered that he could not walk at all because of severe vertigo. This dizziness persisted until two months ago, when it almost completely left him. Following the accident he lost his sense of smell and had a partial loss of taste. About two months after the accident the patient thinks that he recovered some of his hearing in the left ear. He could then understand shouted words. This, however, lasted only a couple of weeks, when he again became totally deaf. A year and a half after the accident he was discharged from the Navy and soon after that noticed that he was regaining some of his hearing. At the present time the patient says he can distinguish high or low tones on the piano and can hear the door-bell. The patient was referred by Dr. Charles W. Burr on March 9, 1915, for an examination of his ears, not only to determine whether his hearing could be restored, but also to determine whether such deafness could be *hysterical* in origin.

We note on the accompanying chart (XX) that high tones are apparently heard in the right ear. With the noise apparatus in the right ear he appears to hear the tuning-fork placed over his left mastoid, so that both cochleas retain some function.

Turning produced no reaction in nystagmus, vertigo or past-pointing, indicating almost a complete destruction of the labyrinths or VIII Nerves.

Douching showed a very marked impairment of responses on both sides. Continuous douching for 3 minutes and 35 seconds on the right side and 2 minutes and

## CHART XIX A

Name James H. Age 38 Date Jan. 7, 1916.

Address

Referred by Dr Chas. H. Frazier

DIAGNOSIS:

SUMMARY:

Complains of Tinnitus

HISTORY:

Dizziness None

Staggering None

Deafness Yes

Tinnitus Yes

NOSE: Negative

THROAT: Negative

EARS: A. D. Negative

A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 2 | A | 10 | Ac | > | Bc | slightly < | n | Pol. | – | $c^4$ | Good | Galt | .7 | Weber |
| | 0 | | 0 | Ac | ? | Bc | ? | n | | – | | 0 | | None | Indefinite |

## CHART XIX B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling None
Romberg Negative
Turning head to right Negative
Turning head to left Negative
Attempt to overthrow Negative

---

### TURNING

To RIGHT →
Amp. Poor
Duration 4 Sec.

To RIGHT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| 12" to Right | 4" to Right |

Nystagmus Very poor
Vertigo Subnormal
Past-pointing Shortened

To LEFT ←
Amp. Large
Duration 13 Sec.

To LEFT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| 24" to Left | 18" to Left |

Nystagmus Fairly good
Vertigo Fairly good
Past-pointing Practically normal

---

### CALORIC

Douche RIGHT ↷
Amp. Good
After 1 min. 15 sec.

Douche RIGHT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| 6" to Right | 3" to Right |

Nystagmus Slightly delayed
Vertigo " "
Past-pointing Normal
Falling Normal

Head Back →
Amp. Good

| RIGHT | LEFT |
|---|---|
| 7" to Right | 5" to Right |

Douche LEFT None
Amp.
After 4 min sec.

Douche LEFT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back None
Amp.

| RIGHT | LEFT |
|---|---|
| Touched | Touched |

## CHART XX A

Name Charles W. C. Age 21 Date March 9, 1915
Address
Referred by Dr Charles W. Burr.

DIAGNOSIS:

SUMMARY:

Complains of Complete deafness and vertigo

HISTORY:
Dizziness None
Staggering Not with eyes open
Deafness Yes
Tinnitus None

NOSE: Hypertrophic rhinitis.

THROAT: Negative. No disturbance of innervation.

EARS:
A. D. Absolutely normal. No evidence of scars.
A. S. Slight impairment of mobility; practically normal

Fistula Negative

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | ? | A | ? | Ac < Bc < n | Pol. | – | $c^4$ | Faint | Galt | ·7 ? | Weber |
| | o | | o | Ac < Bc < n | | – | | 0 | | none | |

Can not understand words when shouted

## CHART XX B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT Slight ↷
Looking UP None
Looking DOWN None

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus Slight, looking to left
Vertigo None
Past-pointing None
Falling None
Romberg Considerable swaying, no special direction
Turning head to right did not increase swaying
Turning head to left " "
Attempt to overthrow normal pelvic movements.

---

### TURNING

To RIGHT None
Amp.
Duration 0 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus Absent
Vertigo Absent
Past-pointing Absent

To LEFT None
Amp.
Duration 0 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 1" to Left | Touched to |

Nystagmus Absent
Vertigo Absent
Past-pointing Absent

---

### CALORIC

Douche RIGHT ↷
Amp. Very small
After 3 min. 35 sec.
Lasted however for 45 seconds.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched to | Touched to |
| | Touched to | Touched to |

Nystagmus Faint but present
Vertigo Absent
Past-pointing Absent
Falling Absent

Head Back
Amp. None

Douche LEFT ↶
Amp. Barely perceptible
After 2 min. 30 sec.
Quickly disappeared

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | ½" to Left | 1½" to Left |
| | Touched to | Touched to |

Nystagmus Almost none
Vertigo Questionable
Past-pointing Present although slight.
Falling Absent

Head Back
Amp. None

30 seconds on the left side produced a slight reaction in nystagmus, showing that some function still remained.

The findings of the vestibular examination in this case would indicate almost a complete destruction of either the VIII Nerves or both labyrinths. In view, however, of the history of the case and the absence of associated paralysis of the VII Nerves or other cranial nerves, the probabilities are that the VIII Nerves are intact, but that the patient sustained a fracture into his labyrinth, the fracture running through the petrous portion of both temporal bones.

The interesting feature about this case was the difficulty, by means of neurologic studies, to exclude hysteria. Because of the absence of any other symptoms except the deafness, hysteria was naturally suspected. It is excluded, however, by the ear-tests, which indicate a definite organic lesion. The tuning-fork tests alone, depending as they do upon the patient's statement whether he hears them or not, can not be definitely relied upon in a differential diagnosis between hysteria and an organic lesion. Generally speaking, we are not yet in a position to state definitely that hysteria could not block the vestibular responses to turning or douching; our belief is that hysteria could not abolish the simple reflex of nystagmus. In this case, however, we have definite evidence of retained function in the vertical semicircular canals of both labyrinths, especially the right, because douching the right ear does produce nystagmus and, although it is a poor reaction, yet it is a reaction all the same. The horizontal canals, on the other hand, show no responses at all. It is inconceivable that a condition like hysteria could affect the cochlear portions of the labyrinths or VIII Nerves, also the fibres from the horizontal semicircular canals and leave a few of the fibres from the vertical semicircular canals still functionating. As near as could be determined by the tests for hearing, the cochlear portions were not completely destroyed. There is no question that both horizontal canals showed no function whatsoever. There is also no question that both vertical semicircular canals of both sides showed some function remaining; such a combination of findings could not be due to hysteria. As this case is not one of hysteria, the prognosis is therefore absolutely bad as regards any return of hearing. This case emphasizes the necessity of a vestibular examination in every case of suspected hysterical deafness.

Case 8.—Mr. Angelo G., age 50. Patient was admitted to the Philadelphia General Hospital on June 10, 1916, complaining of vertigo, staggering and headache.

The history obtained from the family was indefinite; the patient stated that one year before admission he had fallen and struck his head. His family, however, denied this and stated that in September, 1915, the patient had had a sudden chill accompanied by great pain in the head and back of neck. At this time he was first treated for typhoid fever and later a diagnosis was made of "hemorrhage in the brain." He recovered from this acute attack and it was noted that in January, 1916, he was unable to hear in the right ear. Recently he has also lost his hearing in the left ear. Throughout the entire period of the original attack, nine months ago, he had suffered from more or less continuous headache and frequent attacks of vertigo. The patient has a wife and child, in good health.

On admission the patient was scarcely able to walk and reeled towards the left side. He was mentally depressed, reflexes were all exaggerated, clonus was absent and he complained chiefly of severe occipital headache. The provisional neurologic diagnosis was made of lesion of the left cerebellar hemisphere.

Referring to the chart (XXI) of the ear-examination we note that the tuning-fork tests indicate markedly decreased function of each cochlea. The spontaneous nystagmus indicates a disturbance in some portion of the vestibular apparatus. The turning and douching tests show an entire absence of all responses – nystagmus, vertigo, past-pointing and falling—on stimulation of each of the semicircular canals of both ears.

Obviously the otologic diagnosis was a bilateral *peripheral lesion.* There was such a definite absence of *all* responses on stimulation of the vestibular portion of each internal ear: furthermore the marked cochlear impairment corroborated this viewpoint. The following report was made: "The ear examination suggests an almost complete destruction of both labyrinths or VIII Nerves. We believe that the VIII Nerves rather than the labyrinths are involved because of the presence of signs of an accompanying low-grade basilar meningitis (Fig. 114). This viewpoint would also account for the presence of a low-grade fever, which is present in this case. We would suspect a general toxæmia of a virulent type, such as syphilis, and feel that the attack last fall was caused by an involvement more particularly of the right VIII Nerve, and that the present vestibular symptoms have been caused by a recent involvement of the left VIII Nerve."

The Wassermann test, performed subsequently, was reported strongly positive, both for the blood and for the cerebro-spinal fluid. Under anti-syphilitic treatment the patient improved; the staggering, ataxia and headache were relieved, but there was no improvement in the hearing.

Case 9.—Mr. Elberson L., age 52. Patient was admitted to the University Hospital on October 9, 1915, to the service of Dr. William G. Spiller, with the chief complaint of "weakness in the right leg." He gave the following history: Five months before admission he had what he called a "stroke," which was ushered in with nausea and vomiting. The day following the attack he was worse and, in

## CHART XXI A

Name Angelo G. Age 50 Date 6/12/16.
Address
Referred by Philadelphia General Hospital

DIAGNOSIS:

SUMMARY:

Complains of Vertigo, staggering and headache.

HISTORY:
Dizziness Yes
Staggering Yes
Deafness Yes
Tinnitus Yes

NOSE: Negative

THROAT: Negative

EARS:
A. D. Retracted M. T.
A. S. Otitis media, chronic, purulent, healed, but M. T. intact.

Fistula Negative

Hearing Tests

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 1 | A | ? | Ac | > | Bc | < | n | Pol. | | c$^4$ | Fair | Galt | Weber |
| | 0 | | 0 | Ac | > | Bc | < | n | | | | Faint | | + |

## CHART XXI B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

| NYSTAGMUS | | POINTING RIGHT | POINTING LEFT |
|---|---|---|---|
| Looking to RIGHT ⟵ | Shoulder from above | Touched | Touched |
| Looking to LEFT ⟶ | | | |
| Looking UP None | Nystagmus Yes<br>Vertigo Yes | | |
| Looking DOWN None | Past-pointing None<br>Falling to Left | | |
| | Romberg falls to Left | | |
| | Turning head to right<br>Turning head to left<br>Attempt to overthrow } unable to test because of marked ataxia. | | |

### TURNING

| NYSTAGMUS | | POINTING RIGHT | POINTING LEFT |
|---|---|---|---|
| To RIGHT None<br>Amp.<br>Duration Sec. | To RIGHT<br>Shoulder from above | Touched | Touched |
| | Nystagmus None<br>Vertigo None<br>Past-pointing None | | |
| To LEFT None<br>Amp.<br>Duration Sec. | To LEFT<br>Shoulder from above | Touched | Touched |
| | Nystagmus None<br>Vertigo None<br>Past-pointing None | | |

### CALORIC

| NYSTAGMUS | | POINTING RIGHT | POINTING LEFT |
|---|---|---|---|
| Douche RIGHT None<br>Amp.<br>After 5 min. sec. | Douche RIGHT<br>Shoulder from above | Touched | Touched |
| | Nystagmus None<br>Vertigo None<br>Past-pointing None<br>Falling None | | |
| Head Back None<br>Amp. | | Touched | Touched |
| Douche LEFT None<br>Amp.<br>After 5 min. sec. | Douche LEFT<br>Shoulder from above | Touched | Touched |
| | Nystagmus None<br>Vertigo None<br>Past-pointing None<br>Falling None | | |
| Head Back None<br>Amp. | | Touched | Touched |

addition to the nausea and vomiting, he had severe vertigo. On the third day he began to experience difficulty in swallowing. Walking became impossible, so that he had to be taken to the hospital in an ambulance. At this time he had severe shooting pains in the right side of the head, which radiated to the right side of the face and into the right eye. His voice was thick and the difficulty in swallowing became so marked that he had to be fed through a glass tube. There was excessive secretion of saliva. The right leg and foot, he said, were extremely weak, but the condition probably was one of inco-ordination.

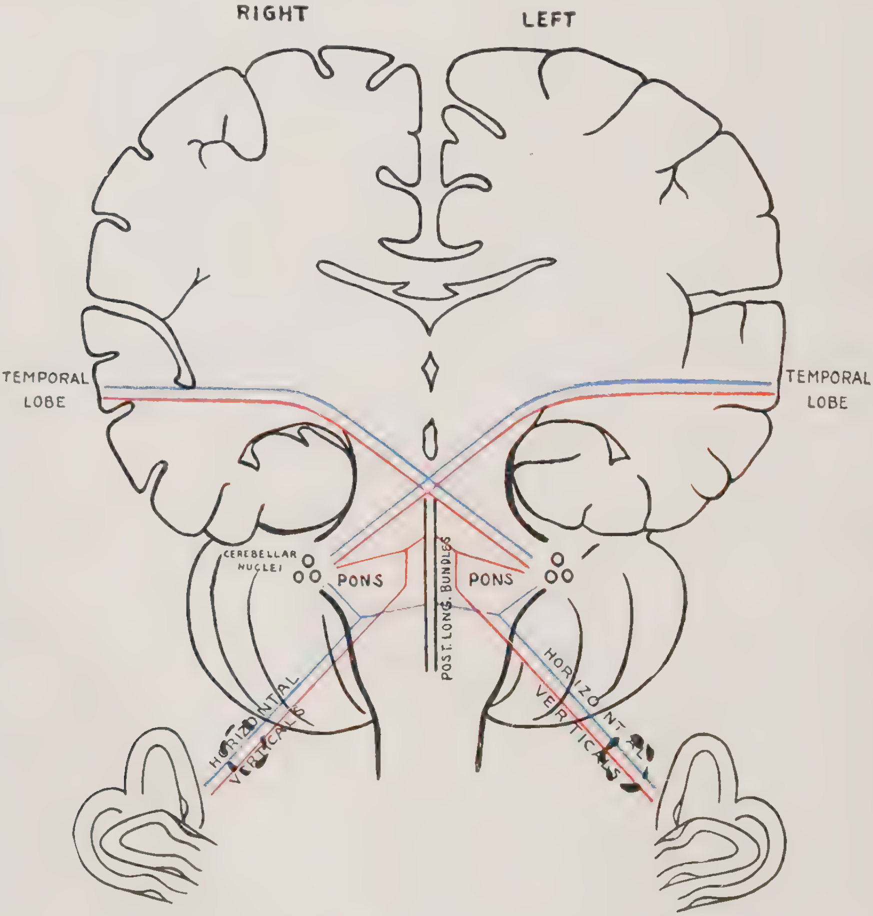

Fig. 114.—Dotted lines indicate location of lesion.

There was nothing of significance in the previous medical or social history. He denied the use of alcohol or tobacco or any history of specific infection. On admission, the following notes of his physical examination were made: "The patient is a well nourished adult, with no evidence of maldevelopment of any kind. Pupils are unequal, the right being smaller than the left; both react normally to light and accommodation. Extra-ocular muscles normal. No facial palsy. Tongue protrudes in midline. On the right side of the face there is loss of sensation for heat, cold and pain, but not for touch. Chest and abdomen negative. There is no weakness of the upper limbs. The reflexes are present in the right upper limb, but diminished in the left. Patellar reflexes are diminished in both lower limbs. No Achilles jerk. No ankle or patellar clonus. When tests for the Babinski sign are made there is a downward motion of the big toe on the right side and no motion in the big toe on the left side."

Notes dictated by Dr. Spiller: "On the left side of face along the edge of the lower jaw in the distribution of the upper cervical nerves, warmth is not as acutely felt as in the corresponding part of the right side of face. In the distribution of the middle branch of the V, the impairment of warmth is distinctly more marked on the right side than on the left side. The impairment of warmth is distinctly greater in the distribution of the first branch of the left V than in that of the second branch. The impairment of pain sensation is very marked in the distribution of the right V and sharply confined to this distribution. Impairment of pain sensation, like that of heat, is distinctly greater in the left upper limb than in the left lower limb, but this impairment is distinct in the lower limb and very distinct in left side of trunk. There is slight impairment of pain sensation in the distribution of upper cervical nerves over the left inferior maxillary bone. There is slight weakness of the right side of soft palate. He still occasionally has difficulty in speaking and swallowing, and at the onset of the disease he was hoarse and spoke in a whisper. Right conjunctival and corneal reflexes greatly impaired—almost lost. Left conjunctival reflex entirely normal. Tactile sense is normal in all parts of the body. No involvement of the tongue. There is numbness in the left side of the body. Finger-to-nose test shows a slight dysmetria, only in the right hand. There is a slight adiadokokinesis in the right hand, both in touching the palm and dorsum of the hand alternately on the right thigh, and in rotating the right hand at the wrist without touching the thigh. Sweeping the upper limb as far back as possible and bringing it back so as to place the finger on the end of the nose, the right upper limb shows marked asynergia. He starts with a sweep of the whole upper limb, but when half completed the movement of the arm is arrested and the sweep is completed by flexion of the forearm. In bending backward as far as he can, he does not flex either knee. The synergic movement of the right lower limb is practically normal."

*Eye report by Dr. Langdon:* "Pupils are unequal, the left being of slightly irregular contour. No synechiæ. Both pupils react to light, accommodation and convergence. There is no loss of power in the orbicularis muscles. There is no hemianopsia and no diplopia could be elicited. The media are clear; the discs, however, are hyperaemic, with blurred nasal margins. The arteries are large and their walls have an increased reflex. There are no hemorrhages, but the picture suggests arterio-sclerosis."

The Wassermann test was negative.

## CHART XXII A

Name Elberson L. Age 53 Date 10/15/15.
Address
Referred by Dr William G. Spiller.

DIAGNOSIS:

SUMMARY:

Complains of Dizziness, staggering and pain in the head.

HISTORY:
Dizziness Has had several attacks previous to "stroke".
Staggering Yes, 4 months.
Deafness None
Tinnitus None

NOSE: Nares rather atrophic

THROAT: Thick pus in both tonsils. On saying "Ah" the uvula is drawn somewhat to the Left. Right vocal cord almost completely paralyzed.

EARS:
A. D. M. T. milky white but mobile.
A. S. M. T. milky white but mobile

Fistula Negative

Hearing Tests

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 3 | A | 9 | Ac | > | Bc | < | n | Pol. | + | $c^4$ | Good | Galt | 1.2 | Weber |
| = | 3 | | 9 | Ac | > | Bc | < | n | | + | | Good. | | 2.6 | ± |

## CHART XXII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None

Looking to LEFT None

Looking UP None

Looking DOWN None

ular muscle coordination
ood. Left pupil larger
han Right.

| | POINTING | |
|---|---|---|
| | RIGHT | LEFT |
| Shoulder from above | Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling there is considerable staggering.
Romberg no swaying
Turning head to right no swaying
Turning head to left causes some swaying forward.
Attempt to overthrow some unsteadiness especially when pushed backward.

### TURNING

To RIGHT ⟶
Amp. Good
Duration 30 Sec.

| | | |
|---|---|---|
| To RIGHT<br>Shoulder from above | 18" to Right | 12" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

To LEFT ⟵
Amp. Good
Duration 26 Sec.

| | | |
|---|---|---|
| To LEFT<br>Shoulder from above | 12" to Left | 24" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing marked

### CALORIC

Douche RIGHT ↷
Amp. Fair
After 1 min. sec.

| | | |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | 4" to Right | 6" to Right |

Nystagmus Normal
Vertigo marked, with nausea and vomiting.
Past-pointing
Falling

| | | |
|---|---|---|
| Head Back ⟶<br>Amp. Large | no vertigo | Touched | Touched |

Douche LEFT ↶
Amp. Good
After min. 20 sec.

| | | |
|---|---|---|
| Douche LEFT<br>Shoulder from above | 8" to Left | 6" to Left |

Nystagmus Good
Vertigo Normal
Past-pointing Normal
Falling Normal

| | | | |
|---|---|---|---|
| Head Back ⟵<br>Amp. Large | Normal vertigo | 4" to Left | 4" to Left |

We have presented the complete neurologic data in this case for a particular reason. Dr. Spiller had made a diagnosis of "Thrombosis of the right posterior inferior cerebellar artery." The patient presented the classical symptom-complex of this lesion; Dr. Spiller, Dr. H. M. Thomas and others have presented identical cases with histologic examination. Dr. Spiller therefore sent the patient for an ear-examination with the following note: "This patient exhibits the definite symptom-complex of a certain lesion in a definite localized area. We have histologic knowledge of identical cases and therefore we can be as sure as is humanly possible of the correctness of the diagnosis. It would therefore be valuable to know what the ear-tests would indicate in this particular case."

The data of the neurologic examination, which we have just presented, were known only to Dr. Spiller, and the ear report was made exclusively on the basis of the ear-examination, by the turning and douching tests.

A glance at the accompanying chart (XXII) would at first seem to indicate that all the responses, both to turning and douching, were normal. There is just one isolated abnormality—douching of the right ear with the head back 60°, although eliciting a normal nystagmus, produced no vertigo and no past-pointing. In other words, the right horizontal canal itself was normal, because it produced a normal nystagmus and yet this stimulation of the right horizontal canal failed to produce vertigo and past-pointing. With this one exception all the responses in nystagmus, vertigo, past-pointing and falling from all the semicircular canals were entirely normal. The labyrinth and VIII Nerves were therefore evidently normal. The pons appeared to be uninvolved, because the responses from the vertical canals of both ears were normal. The cerebellum itself would appear to be unaffected because the vertigo and past-pointing were normal on stimulation of all the semicircular canals except the right horizontal; a normal cerebellum is indicated if stimulation of any canal or canals produces a normal past-pointing of both upper extremities in both directions. A gross lesion of the cerebellum itself will interfere with vertigo and past-pointing no matter what canal is stimulated. With a normal horizontal semicircular canal, normal fibres from the horizontal canal within the VIII Nerve, normal pathways to the posterior longitudinal bundle, resulting in a normal nystagmus, and a normal cerebellum, an interruption of the impulses for vertigo on stimulation of the horizontal canal would therefore suggest a block within the right inferior cerebellar peduncle. The following report was made: "The ear-tests suggest a lesion at the junction of the medulla oblongata and pons on the right side, in the region of the right inferior cerebellar peduncle, not involving the posterior longitudinal bundles." Necropsies obtained in cases of occlusion of the posterior inferior cerebellar artery have shown that the lesion is always situated in this part of the medulla oblongata.

Case 10.—Mr. S. H., age 29. In July, 1914, the patient was seized with a sudden attack of vertigo, staggering, nausea and vomiting. External objects seemed to move around and around in front of him. The diagnosis made was ptomaine poisoning. The patient noticed a distinct improvement every day for three weeks and then things settled down into a *continuous state of dizziness* but without nausea or vomiting. Throughout the attack there was no tinnitus and no impairment of hearing.

*Ear Examination.* Referring to the chart (XXIII), we note a slight rotary nystagmus on looking to the right. Turning with the head upright, testing the horizontal canals, produced distinctly subnormal nystagmus, vertigo and past-pointing. Douching the right ear with the head upright, testing the right vertical canals, produced poor responses; douching the left ear similarly produced impaired responses—in fact, practically no nystagmus, vertigo, past-pointing or falling.

This examination was made over three years prior to this writing. Based on the fact that all responses from all the canals of both ears were impaired, a diagnosis of involvement of both labyrinths was made. Note was made at the time that this diagnosis was not compatible with the normal condition of both cochleas and with certain of the findings by turning and douching. This diagnosis of double peripheral lesion appeared to be corroborated in that subsequent neurologic examination failed to reveal any evidence whatever of intracranial involvement. This diagnosis, however, was not convincing and the patient was advised to report frequently for further tests. Examination a few months later showed practically no nystagmus after turning to the right or to the left with the head upright, and yet, with the head placed 60° backward, turning to the right and to the left produced a large rotary nystagmus. It was obviously impossible to explain this phenomenon on the basis of an end-organ lesion; a report was made that the previous diagnosis of labyrinthitis was apparently wrong and that the case was one of intracranial involvement. Even at that time, however, there was no demonstrable neurologic evidence of an intracranial lesion.

Later there appeared a spontaneous vertical nystagmus, both upward and downward, of the left eye only; also a spontaneous past-pointing of both upper extremities.

The patient at the present time shows unmistakable evidence of an intracranial lesion, most probably a low-grade tubercular basilar meningitis. Corroborating this view, it is to be noted that a gland became greatly enlarged just behind and below the right mastoid; this gland was removed and microscopic examination showed it to be tubercular.

Considering this case from a purely otologic standpoint, a glance at the chart will show that our original diagnosis was not justified. The conclusion of peripheral lesion, based upon an impairment of all the responses to turning and douching, was correct as far as it went; not enough attention, however, was paid to the following data which pointed unmistakably to a central disturbance, even on this first examination:

(1) A spontaneous nystagmus persisted for two months after the original attack and was noted at the first examination.

(2) The continuous spontaneous vertigo was by no means typical of involvement of the labyrinth.

## CHART XXIII A

Name Mr. S. H. Age 29 Date Aug. 28, 1914
Address
Referred by Dr T. H. Weisenburg

DIAGNOSIS:

SUMMARY:

Complains of Vertigo and staggering

HISTORY:
Dizziness Yes
Staggering Yes
Deafness no
Tinnitus no

NOSE: Negative

THROAT: Negative

EARS: A. D. Negative
A. S. Negative

Fistula Negative

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 1½ | A | 6 | Ac > Bc < n | Pol. | – | $c^4$ | Good | Galt | 2.0 | Weber |
| | 1½ | | 6 | Ac > Bc < n | | – | | Good | | 1.4 | ± |

## CHART XXIII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT ↶ Distinct
Looking to LEFT. None
Looking UP None
Looking DOWN None
Looking straight ahead
Slight ↶

Shoulder from above

Nystagmus Yes – to Right.
Vertigo Yes
Past-pointing None
Falling None
Romberg negative
Turning head to right negative
Turning head to left negative
Attempt to overthrow negative

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

### TURNING

To RIGHT →
Amp. Small
Duration 10 Sec.

To RIGHT
Shoulder from above

Nystagmus Impaired
Vertigo Subnormal
Past-pointing Almost none

To LEFT ←
Amp. Fair
Duration 17 Sec.

To LEFT
Shoulder from above

Nystagmus Impaired
Vertigo Subnormal
Past-pointing Almost none

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| To RIGHT | Touched and 2" to Right | Touched and 1" to Right. |
| To LEFT | 1" to Left and Touched | to Touched |

### CALORIC

Douche RIGHT → ↷
Amp. Fair to poor
After 1 min. 20 sec.
but did change
the spontaneous ↶
into ↷
Head Back →
Amp. Poor

Douche RIGHT
Shoulder from above

Nystagmus Impaired
Vertigo Subnormal
Past-pointing Absent
Falling Absent

Douche LEFT ←
Amp. Does not increase
After 2 min. 40 sec. the spontaneous
Left eye shows larger
excursion than right
Head Back
Amp. None

Douche LEFT
Shoulder from above

Nystagmus None practically
Vertigo None
Past-pointing Practically none
Falling Absent.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Douche RIGHT | Touched to | Touched to |
| Head Back | 2" to Right and Touched | 1" to Right |
| Douche LEFT | Touched to | 1" to Left |
| Head Back | Touched to | Touched to |

(3) Douching the right vertical canals produced a *horizontal* nystagmus as well as a rotary nystagmus; this *perverted* nystagmus directly indicated intracranial involvement and should have pointed the way to a correct diagnosis.

We were evidently dealing in this case with a lesion which was both peripheral and central, and was therefore most probably so located intracranially that it was in a position to involve both VIII Nerves. If our original diagnosis had been a neuritis of both VIII Nerves, instead of a labyrinthitis, it would have been true although only a half-truth. As it was, our original conclusions were altogether erroneous and misleading; it was only after subsequent examinations that the ear tests made a correct diagnosis of intracranial involvement. The fault at the time of the first examination was not in the data shown by the tests, but in our inability to understand and interpret the phenomena which we now see were plainly presented.

Case 11.—Mrs. C. M. D., age 36, was admitted to the University Hospital to the services of Drs. Mills and Spiller August 12, 1915, having been referred by Drs. S. D. Ludlum and J. W. McConnell. Her chief complaint was vertigo and inability to walk. She had apparently been in good health until eight weeks before her admission to the hospital, and she died two weeks after admission; so that her entire illness was of only ten weeks' duration.

She gave the following history: The trouble began with vertigo. Two weeks after the onset the vertigo was so marked that she was unable to walk; she staggered both to the right and to the left. At this time she began to have vomiting spells, without precedent nausea; these spells continued at intervals until her death. During the fourth week of her illness the patient noticed that the right limbs were becoming weak; this impairment steadily increased until at the time of her admission to the hospital she could scarcely move either the right upper or lower extremities. Five weeks from the time of onset she began to have severe headaches and in the sixth week she noticed that her vision was failing. No symptoms referable to the pulmonary, cardiac or renal systems. Family history negative, except that one uncle died of brain tumor.

The following are the notes of Dr. Mills' examination, made immediately after her admission: "The patient lies in bed in the supine position, with the head slightly inclined to the left. She has a typical right-sided hemiplegia of the face, arm and leg, with moderate hypertonicity; superficial and deep reflexes are all increased, foot clonus is absent and plantar stimulation gives plantar flexion. No paralysis is discoverable on the left side, but the left finger-to-nose test shows a distinct ataxia, the uncertainty being of small excursion. Sensation for pain and touch is everywhere normal. Speech is slow and dysarthric, but not sufficiently so to make it inarticulate; she answers questions sensibly and obeys commands, in so far as she is physically able. The right eye shows slight ptosis; there is some weakness of the external rectus, the internal rectus and the superior and inferior obliques. The left eye shows ptosis and weakness in all ocular movements; movements of the musculature supplied by the III Nerve are almost completely abolished. Conjugate movements are lost in all directions. The pupils are unequal, the right being larger. The pupils react to direct light stimulation, but the convergence response is questionable."

Dr. B. F. Baer, Jr., reported marked papillœdema, worse in the left eye.

The ear examination was made immediately after admission and the findings are shown on the accompanying chart (XXIV). The following deviations from the normal are to be noted:

(1) There is a spontaneous nystagmus in every direction; the vertical nystagmus upward is the most significant, because it is suggestive of an involvement of the brain-stem.

(2) Turning to the right produced a *rotary* nystagmus to the left instead of a horizontal nystagmus; this "perverted" nystagmus is also indicative of a brain-stem lesion.

(3) Turning to the right and to the left produced a distinct eye movement, but no vertigo whatever. This, *per se,* would suggest an interference with the pathways from both horizontal canals to the cerebral cortex, at the point of the decussation of the superior cerebellar peduncles in the base of the cerebral crura.

(4) Douching the right ear, with the head 30° forward, which stimulates the right vertical canals, fails to produce any responses whatever. This suggests involvement of the vertical canals fibres in the right side of the pons.

(5) Douching the right ear with the head back 60°, stimulating the right horizontal canal, on the other hand, does produce a movement of the eyes; this would indicate normal pathways in the medulla oblongata.

(6) It must be noted that turning and douching would for the most part produce a *conjugate deviation* of the eyes instead of a nystagmus; the only instance in which there was a real nystagmus was after turning to the right. This interference with almost all of the impulses which would normally cause the quick recoil of the eyes—the cerebral component which completes a real nystagmus—would indicate a lesion at the junction of the cerebral crura at the point where the cerebro-ocular fibres from both sides of the cerebral cortex, come together to be distributed to the eye-muscle nuclei.

(7) There is a striking absence of *vertigo;* no vertigo whatever was produced by stimulation of any canal except the left vertical canals, which did produce a noticeable vertigo. Even this vertigo, however, was very slight; the patient merely stated "that made me a little dizzy."

*Analysis.*—Both labyrinths and both VIII Nerves are apparently normal, because both cochleas were normal and stimulation of each ear produced *some* normal responses. The medulla oblongata appeared to be normal because stimulation of the horizontal canal of each ear produced a normal eye-movement; the conjugate devia-

## CHART XXIV A

Name Mrs C. M. D. Age 36 Date 8/13/15.

Address

Referred by Dr Mills and Dr Spiller.

DIAGNOSIS:

SUMMARY:

Complains of Vertigo and weakness.

HISTORY:

Dizziness Yes

Staggering Yes

Deafness None

Tinnitus None

NOSE: Negative

THROAT: Negative

EARS:

A. D. Negative

A. S. Negative

Fistula, Negative

Hearing Tests

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 2 | A | 7 | Ac > Bc = n | Pol. | | $c^4$ | Good | Galt | .9 |
| | 2 | | 7 | Ac > Bc = n | | | | Good | | .9 |

## CHART XXIV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT ← marked
Looking to LEFT → marked
Looking UP ↑ occasional
Looking DOWN
Paresis Right internal rectus
Ptosis Left upper lid

| POINTING | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Unable to lift Right arm. | 4° to Left |

Nystagmus Yes
Vertigo Yes
Past-pointing Yes
Falling Yes
Romberg —
Turning head to right —
Turning head to left —
Attempt to overthrow Impossible to make tests.

---

### TURNING

To RIGHT ↷
Amp. Good
Duration 34 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | to<br>Impossible | to<br>✓ |

Nystagmus Exaggerated and perverted
Vertigo None
Past-pointing —

To LEFT ←
Amp.
Duration ? Sec.
Mostly a conjugate deviation to Left.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | to<br>✓ | to<br>✓ |

Nystagmus
Vertigo
Past-pointing

---

### CALORIC

Douche RIGHT None
Amp.
After 4 min. sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | to<br>✓ | to<br>✓ |

Nystagmus Absent
Vertigo Absent
Past-pointing —
Falling Absent

| | | |
|---|---|---|
| Head Back Vertigo absent<br>Amp. Mostly conjugate deviation, marked | to<br>✓ | to<br>✓ |

Douche LEFT Conjugate deviation
Amp. to Left.
After min. 40 sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | to<br>✓ | to<br>✓ |

Nystagmus
Vertigo
Past-pointing
Falling

| | | |
|---|---|---|
| Head Back<br>Amp. Conjugate deviation to Left. | to<br>✓ | to<br>✓ |

tion which was produced was a perfectly normal response so far as the stimulation from the horizontal canal itself was concerned. The absence of the quick component of the nystagmus would indicate a supranuclear interference. The striking phenomena of this examination were the absence of vertigo and the absence of the quick component of nystagmus; *both* of these phenomena indicate a lesion *at the same point*—the base of the cerebral crura. The impairment of vertigo from stimulation of all the canals would indicate a lesion at the decussation of the superior cerebellar peduncles in the base of the cerebral crura and the impairment of the quick component of nystagmus would similarly indicate a lesion at the same point because of an interference with the impulses coming from the cerebrum to the eye-

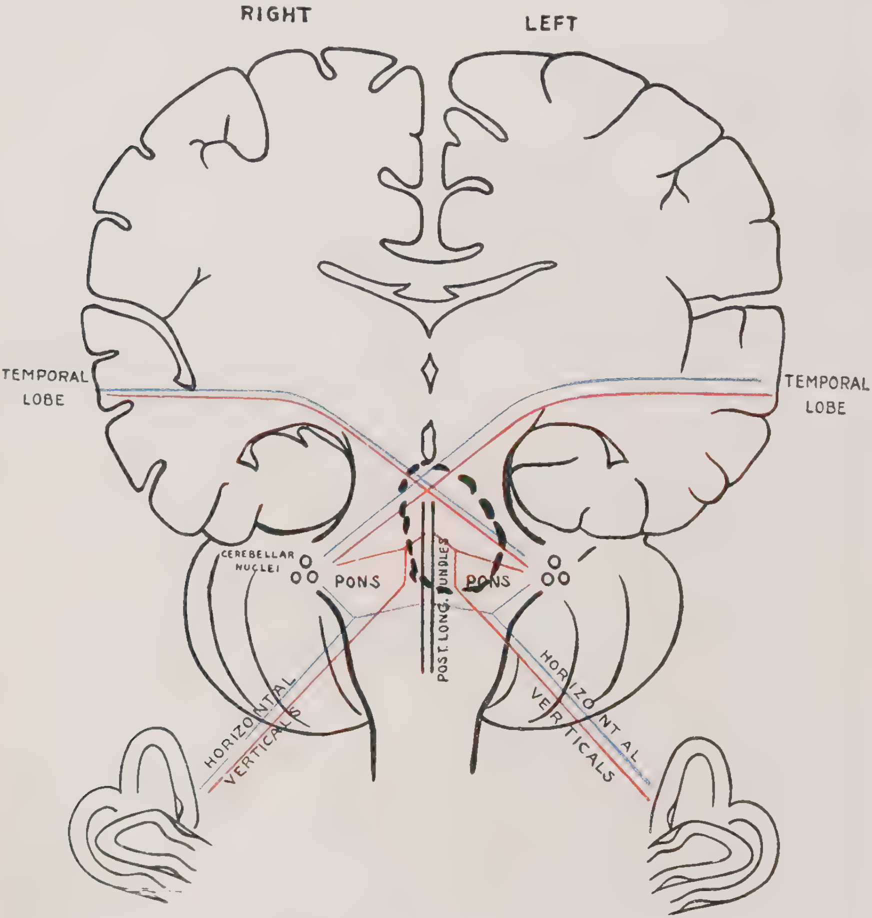

Fig. 115.—Dotted lines indicate location of lesion.

muscle nuclei. The following report was made in writing: "The results of the ear examination suggest a lesion of the upper portion of the pons at the junction of the cerebral crura at the point where the cerebral fibres reach the region of the posterior longitudinal bundles (Fig. 115). The medulla oblongata and the lower half of the pons appear uninvolved."

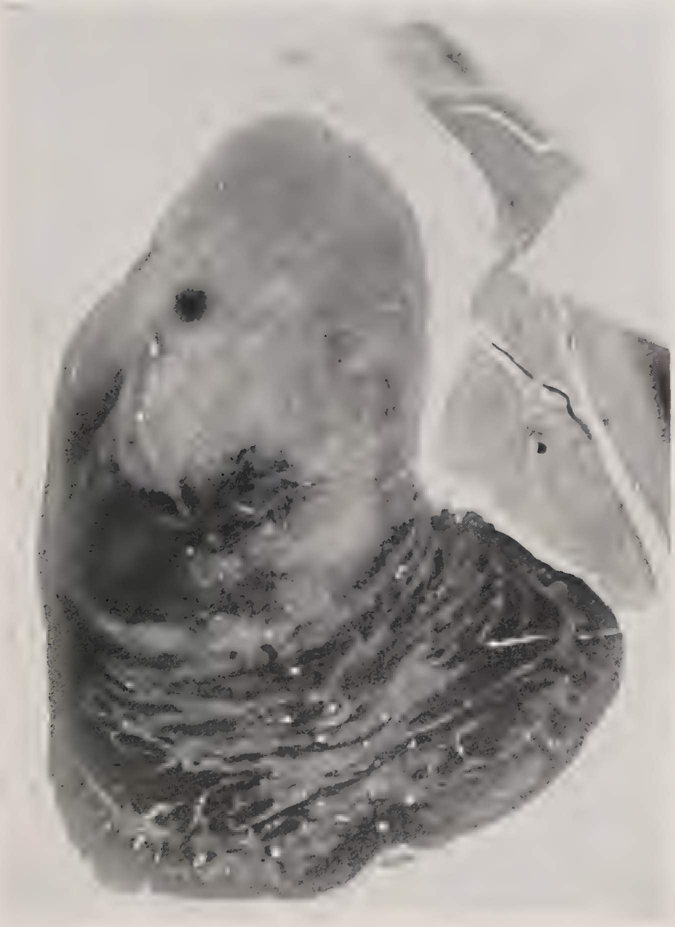

Fig. 116. (Case 11.) Glioma of the pons.

*Autopsy.*—Dr. William G. Spiller made the following report of his macroscopic and microscopic examination:

"The tumor extends from the uppermost portion of the cerebral peduncles, through the pons, and into the left cerebellar lobe. It is confined to the left half of the brain-stem throughout. It causes much enlargement of the left side of the cerebral peduncles, and implicates almost all of this side. In the upper part of the pons the posterior longitudinal bundle on the right side is well preserved, but the outer portion of the posterior longitudinal bundle on the left side is much degenerated. The tumor in the upper part of the pons is confined to the left tegmentum. The same is true of the middle portion of the pons. The tumor greatly compresses the IV ventricle and almost closes it by displacement of the surrounding tissue. It does not invade the medulla oblongata, but at the level of this structure it is confined to the white matter of the left cerebellar lobe. It is a glioma (Fig. 116)."

### Comment

A study of the data obtained by the ear-tests and necropsy indicates certain definite facts, as to the vestibular pathways in the brain-stem. The examination demonstrated that the tracts from all the semicircular canals except the right verticals, through the vestibular nuclei to the nuclei of the ocular nerves were open. The specimen showed that the Deiters' nucleus group (Deiters' nucleus proper, the triangular nucleus and von Bechterew's nucleus) was unaffected by the lesion, as were also the entire medulla oblongata and the lower part of the pons.

Let us recall the peculiar response of conjugate deviation. This ocular movement may be regarded as the slow component of nystagmus. For it to occur it is only necessary that the stimuli shall pass from the ear through the posterior longitudinal bundle to the nuclei of the VI Nerve and of the III Nerve (or rather of that part of this nerve which innervates the internal rectus). The conjugate deviation, therefore, results from stimulation of the ear; but this normally is converted into a nystagmus by impulses from the cerebrum which tend to call the eye back to its usual position, that is, if the cerebro-oculo-nuclear tracts are open. It follows, therefore, that if, as in this case, the response is only a conjugate deviation, some lesion is interfering with the transmission of the cerebral stimuli to the nuclei of the ocular nerves affected. It has been shown by necropsy that such a lesion was present.

It may be of interest to note in this connection that one fully anesthetized, when the semicircular canals are stimulated, shows only a conjugate deviation and no return movement.

Let us turn now to an explanation of the vertigo in the light of our premortem and postmortem findings. We believe that the ear stimulus which produces vertigo passes to the cerebrum through the *cerebellum.* While the paths which carry the vestibular stimuli through the cerebellum to the cerebrum are not demonstrated absolutely in all their extent, the facts at our disposal appear to indicate that they are received by the cerebellum through the inferior cerebellar peduncle from the horizontal canals and the middle cerebellar peduncle from the vertical canals, and after completing their cerebellar itinerary, pass to the cerebrum by way of the superior cerebellar peduncles. In the present case, ear-stimulation failed to produce the normal vertigo from all the semicircular canals except the left verticals. Clearly, therefore, there was obstruction somewhere in this vestibulo-cerebello-cerebral pathway concerned with the stimuli causing vertigo. As there was present a conjugate deviation from the stimulation of the same canals or tracts, the lower neuraxial pathways appeared open. To explain this absence of vertigo the logical place of destruction was at the decussation of the superior cerebellar peduncles. The necropsy studies showed involvement of the superior cerebellar peduncles, which have been demonstrated to contain tracts which pass to the cerebrum from the cerebellum.

With regard to the spontaneous nystagmus upward, this case confirms our previous experience that such a nystagmus is indicative of a brain-stem lesion, either destructive or due to pressure.

The right vertical canals failed to produce any reactions. It follows, therefore, that the neuraxial fibres from these canals were involved, and as the lesion was in the upper portion of the pons, that these fibres must reach at least as high as the site of this lesion. That the lesion extended further down on the left side suggests that the fibres from the right vertical canals may decussate to the opposite side.

Case 12.—Mr. C. S., age 56. The patient was admitted to the Hahnemann Hospital October 1, 1916, complaining of vertigo, staggering, projectile vomiting, severe headache and weakness of the lower extremities, especially the left.

## CHART XXV A

Name *C. S.* Age *56* Date *Oct. 27, 1916.*
Address *Hahnemann Hospital*
Referred by *Dr Palen, Dr. Bartlett & Dr. Clay.*

DIAGNOSIS:

SUMMARY:

Complains of *Vertigo, staggering and headache*

HISTORY:
Dizziness *Yes*
Staggering *Yes*
Deafness *No*
Tinnitus *No*

NOSE: *Negative*

THROAT: *Negative*

EARS:
A. D. *Slight retraction, otherwise normal*
A. S. *Slight retraction, otherwise normal*

Fistula *Negative*

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| A | *1* | A | *7* | Ac *>* Bc = n | Pol. | | $c^4$ | *Good.* | Galt | *.7* | *Webe* |
| = | *1.* | | *7* | Ac *>* Bc = n | . | | | *Good* | | *.7* | *±* |

## CHART XXV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None.

Shoulder from above

Nystagmus None
Vertigo Yes
Past-pointing None
Falling
Romberg falls backward
Turning head to right not tested
Turning head to left " "
Attempt to overthrow " "

POINTING

| RIGHT | LEFT |
|---|---|
| Touched<br>Incoordinate | Touched<br>Incoordinate. |

### TURNING

endency to conjugate deviation to
ght - slightly oscillatory

To RIGHT →
Amp. Unusually large
Duration 33 Sec.

To RIGHT
Shoulder from above "

Nystagmus exaggerated
Vertigo Normal
Past-pointing exaggerated

| RIGHT | LEFT |
|---|---|
| 36" to Right<br>20" to Right | 8" to Right- |

To LEFT ←
Amp. Unusually large
Duration 35 Sec.
endency to conjugate
eviation to Left

To LEFT
Shoulder from above

Nystagmus exaggerated
Vertigo Normal
Past-pointing exaggerated

| RIGHT | LEFT |
|---|---|
| 36" to Left<br>18" to Left | 36" to Left<br>12" to Left |

### CALORIC

Douche RIGHT None
Amp.
After 4 min. sec.

Douche RIGHT
Shoulder from above

Nystagmus Absent
Vertigo Subnormal
Past-pointing "
Falling "

| RIGHT | LEFT |
|---|---|
| Touched to 2" to Right | 6" to Right. |

Head Back →
Amp. Good
Head forward

| RIGHT | LEFT |
|---|---|
| Touched | 10" to Right |
| 6" to Left | Touched |

Douche LEFT None
Amp.
After 4 min. sec.

Douche LEFT
Shoulder from above

Nystagmus Absent
Vertigo Subnormal
Past-pointing "
Falling "

| RIGHT | LEFT |
|---|---|
| 4" to Left | Touched |

Head Back ←
Amp. Good
Head forward

| RIGHT | LEFT |
|---|---|
| 24" to Left | 24" to Left |
| 24" to Right | 6" to Right- |

From Dr. I. H. Jones' Equilibrium and Vertigo.

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He was in good health until four years before admission, at which time he noticed that it was becoming difficult for him to walk in a straight line. At about the same time he began to have "dizzy spells." As time progressed he found that while walking he always veered toward the left. This inability to walk straight and the occasional attacks of vertigo were the only symptoms for the first year of his illness. Then, three years ago, he began to have the severe headaches which have been present almost constantly ever since, and at the same time he noticed that he was getting blind. One year before admission the attacks of vertigo became much more frequent and the staggering became so severe that frequently he would be unable to stand upright without grasping some object for support.

The family history and previous medical history were negative. The following notes of his physical examination were made at the hospital: "Drooping of the left side of the face. The grip is poor in each hand and there is considerable incoordination of both upper extremities. Loss of power in both lower extremities, more marked in the left, and marked ataxia with a tendency to falling, especially backward. The patellar reflexes are normal. Pupils react normally to light and accommodation. Examination of the eye-grounds shows double choked disc with dilated blood-vessels. The visual fields are concentrically contracted. The patient's mentality is poor; he answers questions slowly and does not even know what year it is."

The first ear-examination was made on October 16, 1916, by Drs. Palen and Clay. The cochleas were found to be normal: the only abnormality in the hearing was a slight diminution for the lower tones. Turning to the right and the left each produced a nystagmus of 35 seconds' duration; this showed that the horizontal canals were not only functionating, but produced hyperactive responses. Douching the right ear produced responses after one minute and thirty seconds of douching. Douching the left ear produced responses after one minute and twenty seconds. This showed that the responses from the vertical canals of both sides were present, although somewhat delayed.

The patient was examined by us only nine days later and yet, as the accompanying chart (XXV) shows, there had occurred marked changes in the vestibular mechanism in that short time.

In interpreting the findings on the chart, it is evident at a glance that we are dealing with a central lesion. The vertical canals of both ears show no responses, whereas the horizontal canal of each ear shows hyperactive responses both to turning and douching. The right cochlea is normal, the right horizontal canal is functionating and yet the right vertical canals produce no responses. The same phenomenon-complex is shown by the left ear. Therefore it would be highly improbable that the vertical canals of each ear had become destroyed and it would be more reasonable to assume that the vertical canals fibres had become blocked intracranially. This view is corroborated in that after turning to the right, instead of a clean-cut nystagmus to the left, there is a tendency to conjugate deviation to the right; similarly after turning to the left there is a tendency to conjugate deviation to the left. So far, therefore, the tests suggest that the lesion is central.

A more exact location of the lesion is best attained by first eliminating the normal portions of the vestibular apparatus:

(1) The labyrinths and VIII Nerves are normal, because the hearing is unimpaired and the horizontal canals produce good responses.

(2) The medulla oblongata would appear normal because when each horizontal canal is tested separately, by putting the head back after douching, normal responses appear.

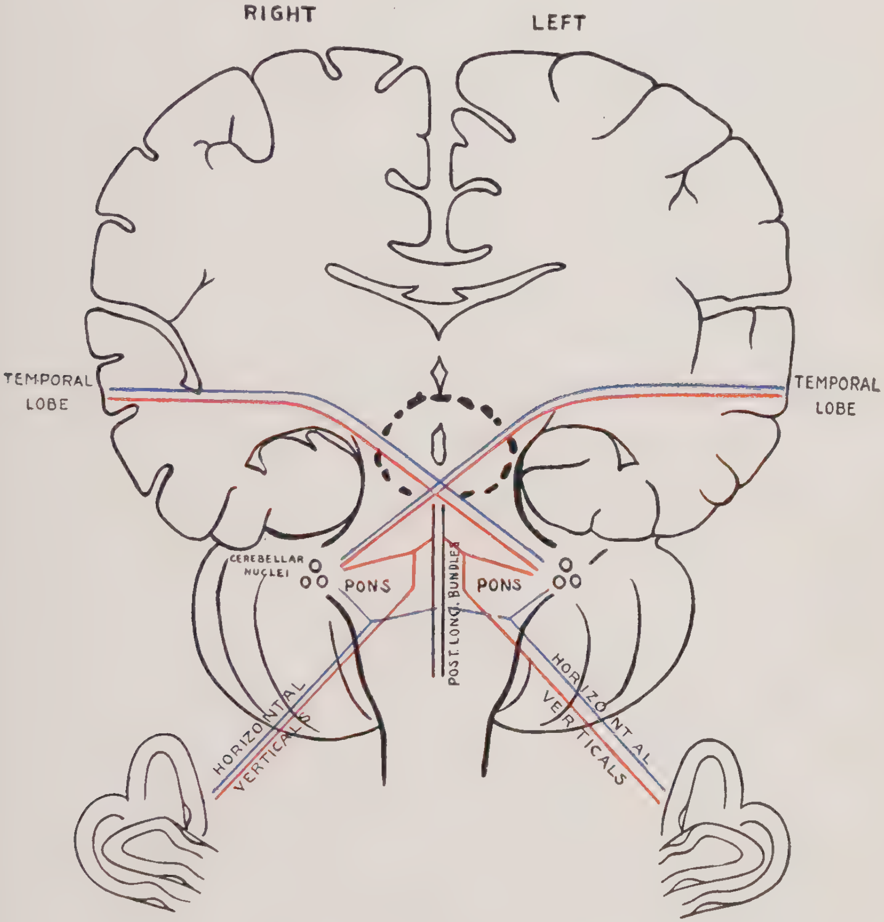


Fig. 117.—Dotted line indicates location of lesion.

(3) The cerebellar cortex would appear to be normal because we were able to elicit a normal past-pointing of both arms in both directions.

If then we consider that the labyrinths, VIII Nerves, medulla oblongata and cerebellar cortex are uninvolved, it would at first seem reasonable to decide that the vertical canals fibres were involved in the pons. This, however, would not seem likely because when the patient was examined only nine days previously reactions from the vertical canals of both ears *were* obtained. It would be improbable that a lesion should extend so far *into both sides of the pons* within these nine days; a more simple explanation of this phenomenon would be that there had occurred an increase of pressure in this region. The tendency to a conjugate deviation both to the right and to the left indicates an interference with the cerebro-ocular fibres at the base of the cerebral crura, where the fibres from the cerebral hemispheres to the eye-muscle nuclei enter the posterior longitudinal bundles.

The following report was therefore made:

"The ear-tests suggest a tumor at the junction of the anterior superior aspect of the cerebellum and the brain-stem (Fig. 117). Speaking purely from the standpoint of the ear-tests, the tumor would appear to be centrally located and therefore inoperable. There is no evidence to suggest that one side is more involved than the other. The following appear normal: (1) Labyrinths, (2) VIII Nerves, (3) Medulla oblongata, (4) Posterior longitudinal bundles, (5) Inferior cerebellar peduncles, (6) Cerebellar cortex throughout."

Report of autopsy by Doctor Sappington: "The site of the corpora quadrigemina was occupied by an oval tumor three inches by an inch and a half in size. The corpora quadrigemina themselves were not in evidence, apparently being obliterated by the growth. The tumor was situated in the middle line just posterior to the posterior commissure and pineal body and just anterior to the velum medularæ anterius and also just anterior to the origin of the trochlear nerves. The tumor practically roofs the aqueduct of Sylvius. There were no gross hemorrhages in or about the tumor. The tumor itself was about the firmness of the cerebral tissue and on section appeared whitish gray and in some places was almost caseous. The lateral, the third and the fourth ventricles show no gross changes. Cerebrum normal. Section of the cerebellum showed it to be normal. Medulla oblongata, pons, inferior and middle cerebellar peduncles were also normal."

We note that the tumor was in the exact spot suggested by the ear-tests.

Case 13.—Mr. E. B., age 24. This patient was a college student, apparently in excellent health, and merely complaining of slight attacks of dizziness. He suffered very little inconvenience and regarded the matter lightly; it was only on plying him with questions that the following details were elicited: One month before the ear examination he noticed that he had some difficulty in keeping step while he was dancing; this inco-ordination seemed to be most marked when he would be looking at the other dancers. He also recalled that, at about the same time, if he moved his head quickly, it would make him dizzy. A week later he began to stagger a little; when it first came on, it was so slight that he was never aware of it himself, but was told of it by other people. Soon, however, it became so marked that very frequently he would have to hold on to objects for support. During these attacks of staggering he would lean to the right and forward and had a sensation of some heavy weight on his head and shoulders. At no time did he have much tinnitus; only once, one week before this examination, he noted a slight singing in the ears.

The results of the ear-examination are given on the accompanying chart

(XXVI). In attempting an explanation of the findings, the following deductions were made:

(1) We are dealing with an organic lesion and not with a functional neurosis. The accompanying vestibular chart shows definitely impaired responses—the result of a damaged ear-mechanism.

(2) Having determined that there is an organic lesion involving the ear-mechanism, we are led to believe that this lesion is central and not peripheral for the following reasons: (*a*) Perfectly good hearing on both sides. With intact cochleas it is fair to assume that the internal ears in their entirety are unaffected. (*b*) The presence of a spontaneous vertical nystagmus, both upward and downward; this is pathognomonic of a central lesion and would indicate a brain-stem disturbance. (*c*) There appears to be a difference in the excursion of the eyes when looking to the left; this inco-ordination of ocular movement obviously could not be produced by disturbance in the labyrinths. (*d*) Both the turning and the douching produced a fair nystagmus, but the past-pointing for one or the other arm was very poor, almost absent or in the wrong direction. Such a differentiation must, of course, be central and not peripheral.

(3) There is no one lesion that could account for all of these disturbances. The examination would therefore suggest multiple lesions such as would occur in a case of disseminated sclerosis.

A study of the responses to the pointing tests shows that there is an impairment in the pointing of each arm outward. This was so constant that it made probable an actual small lesion of the cerebellum itself, rather than in the pathways on the way to the cerebellum. The nystagmus produced by stimulating the semicircular canals was abnormal, but the degree of impairment was different or varied with each canal stimulated; this would suggest multiple lesions in the brain-stem. There is no evidence of any large lesion anywhere, since not a single response is completely abolished.

Several months later, July 21, 1916, this patient was admitted to the Ophthalmologic Department of the University Hospital because of failing vision. He improved somewhat and left the hospital. Two months later, September 21, 1916, he was readmitted—this time to the neurologic service. He had suffered a sudden change for the worse; at this time he was hardly able to walk. His vertigo was very severe, the weakness in the legs was getting gradually worse, his speech had become slurring, nasal and monotonous, and he became emaciated and weak. The following notes were made by Dr. William G. Spiller: "Pupils equal and react equally and promptly to light, accommodation and convergence. Facial musculature unimpaired. The tongue is protruded slightly to the right of the midline and is freely movable to either side.

"*Extremities.*—Grip is fair with the right hand, poor with the left. There is a marked ataxia of the upper extremities and at times there develops a gross intention tremor in both upper extremities on attempting the finger-to-nose test and the finger-to-finger test. Adiadokokinesis is present in both forearms. Sense of position and stereognostic sense are normal in both hands. Biceps jerk normal and equal on both sides; triceps jerk questionably present on both sides. Knee jerk slightly exaggerated on the right side, normal on the left. Achilles jerk mod-

## CHART XXVI A

Name Mr. E. B. Age 24 Date 3/3/1916.
Address
Referred by Dr J. V. C. Clay.

DIAGNOSIS:

SUMMARY:

Complains of Vertigo and staggering, slight.

HISTORY:
Dizziness At times, started 1 month ago.
Staggering At times, started 3 weeks ago.
Deafness None
Tinnitus None

NOSE: Negative

THROAT: Negative

EARS:
A. D. Slight retraction, otherwise negative.
A. S. Slight retraction, otherwise negative.

Fistula Negative

Hearing Tests

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 1/2 | A | 8 | Ac | > | Bc | < | n | Pol. | | $c^4$ | Good | Galt | Weber |
| = | 1/2 | | 8 | Ac | > | Bc | < | n | | | | Good | | ± |

## CHART XXVI B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT ← marked, coarse, slow
Looking to LEFT → marked, rapid
Looking UP ↑ marked
Looking DOWN ↓ marked

Weakness of left ext. rectus

| POINTING | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus in every direction
Vertigo none
Past-pointing none
Falling none
Romberg sways with eyes open
Turning head to right does not increase swaying
Turning head to left " " " "
Attempt to overthrow not tested

### TURNING

To RIGHT →
Amp. Poor. Irregular
Duration 14 Sec.

Tendency to conjugate deviation

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 5" to Right<br>and touched | 6" to Right.<br>6" " Right<br>5" " Right<br>6" " Right<br>Continues long time |

Nystagmus Irregular
Vertigo Normal
Past-pointing Abnormal

To LEFT ←
Amp. Good - Irregular.
Duration 28 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 6" to Left<br>6" Left<br>5" Left | 1" to Left<br>1" Left<br>and touched |

Nystagmus Irregular
Vertigo Normal
Past-pointing Abnormal

### CALORIC

Douche RIGHT ↷
Amp. Good
After 2 min. 15 sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | 2" to Right<br>and touched |
| Head Back | 6" to Left | 3" to Left<br>and touched |

Nystagmus Good
Vertigo Markedly impaired
Past-pointing markedly impaired
Falling Absent

Head Back Tendency to conjugate deviation
Amp.

Douche LEFT ↶
Amp. Fair
After 1 min. 30 sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 8" to Left | 8" to Left |
| Head Back | 8" to Left | 6" to Right |

Nystagmus Fair
Vertigo Present
Past-pointing Normal
Falling Present

Head Back ←
Amp. Good

erate and equal on both sides. Ankle-clonus obtained on the right side on the first examination, but cannot be elicited on either side on the second examination. Heel-to-knee test shows marked ataxia on both sides. Both lower extremities and trunk are ataxic. He cannot stand or even sit up unsupported. There is more power in extension of the knees than in flexion. There are frequent and rapid changes in amount of power of all the movements of the extremities. Sense of touch and pain normal throughout. Cremasteric reflex prompt on the left side, but sluggish on the right.

*Eye examination by Dr. de Schweinitz* on September 26, 1916, showed a marked paling of the upper half of the optic nerve on each side and a paralysis of the right internal rectus.

A Wassermann of the blood was repeatedly negative.

An examination of the urine, the lungs and heart was negative.

On October 10, 1916, it was noted that the patient was a little flighty and had delusions.

He died suddenly on December 21, 1916.

Autopsy by Dr. William G. Spiller: "Brain substance has a firm, dense feel. Arteries of the base show no fibrosis. The lateral ventricles are distended and remain rigidly extended even when incised, suggesting a special firmness of the surrounding brain substance." Histologic examination showed typical changes of multiple sclerosis throughout brain and spinal cord.

### COMMENT

It is to be noted that when this young student first presented himself he was in excellent general health, except for slight vertigo. It is from such cases as this that we learn how serious may be the significance of even a slight dizziness; this case also emphasizes that regardless of the general health of the patient, a well-developed spontaneous vertical nystagmus indicates a serious intracranial disturbance and the prognosis in such conditions should always be guarded. At the time of the original ear-examination, we were so impressed with the serious possibilities in this case that moving pictures were taken of his responses to ear-stimulation as well as of the spontaneous vertical nystagmus upward and downward. It is important to note that the ear-examination in this case gave definite information in regard to the nature of his disease very early in its development. The autopsy corroborated the evidence furnished by the original ear-examination.

CASE 14.—Stephen F., age 12. This patient was referred for an ear examination with a provisional diagnosis of cerebellar tumor. The patient was admitted to the hospital of the University of Pennsylvania on November 4, 1914, with the chief complaint of headache, vomiting and fever.

Patient was well until one month before admission, when he began to have headache, fever and vomiting. The patient has not vomited within the last week. During the first week of his illness he had a slight rise of temperature and was markedly prostrated. No convulsions or diarrhœa. During the past week there was an evening rise of temperature associated with sweats. The headaches were always worse at night. Patient cries with pain in his neck and side.

Physical examination. Slight tache and Kernig's present. Spinal fluid clear, but under great pressure. Patient has a tendency to walk to the left and carries his head to the left. Biceps jerks very slight. Knee jerks absent. Achilles prompt. No Babinski or ankle clonus.

On the day following admission to the hospital the boy vomited several times in the morning; at 11 A.M. he had a peculiar attack, during which, he later said, he was blind. There was flexion of the arms, hands were flexed at the wrist and the fingers held stiff and drawn together. This convulsive seizure disappeared promptly on the right side, but persisted for 45 minutes on the left side. During the attack he vomited and complained of severe headache. A lumbar puncture taken during the attack showed the spinal fluid under great pressure. Patient was examined by a neurologist the day following, who reported as follows: "Adiadokokinesis more marked in the left hand; in the finger-to-nose test the left hand overshoots; left heel-to-heel test shows some awkwardness. Towards the end of a general movement the patient executes two or three movements of small amplitude. Loss of knee jerks. Achilles present. Disturbance of associated ocular movements to left. In walking patient has a tendency to lean forward and to left. Subtentorial decompression advised."

Two weeks later patient developed a partial paralysis of the left external rectus, also marked rigidity of the neck.

Eye-examination revealed a high grade choked disc in both eyes.

Wassermann reaction was negative.

The first examination of his vestibular apparatus was made on November 4, 1914, and the findings recorded on the accompanying chart (XXVII).

It will be noted on the chart that the hearing of both ears was entirely normal and that the vestibular apparatus responded quickly and correctly to all the tests. The nystagmus was of good amplitude and appeared promptly after both turning and douching; the past-pointing also after each form of ear-stimulation was normal. The ear report was: "The normal responses in nystagmus, vertigo, past-pointing and falling, after the turning and douching tests, would suggest normal labyrinths, VIII Nerves, medulla oblongata, pons and *cerebellum.*"

Two weeks later another ear-examination was made, which showed startling changes in the responses (Chart XXVIII). Just as before, the patient showed a normal nystagmus and past-pointing after turning, but douching each ear with the head upright, stimulating the vertical canals, produced no responses whatever. The douching was continuous for 4 minutes in the right ear and for 5 minutes in the left. After douching the left ear with the head upright, the patient was taken from the chair and put back on the stretcher. Immediately the patient exclaimed

## CHART XXVII A

Name Stephen F. Age 12 Date Nov. 4, 1914
Address
Referred by University Hospital.

DIAGNOSIS:

SUMMARY:

Complains of Headache, vertigo, vomiting dimness of vision.

HISTORY:
Dizziness Yes
Staggering Yes
Deafness No
Tinnitus No

NOSE: Negative

THROAT: Negative

EARS:
A. D. Normal
A. S. Normal

Fistula Negative

Hearing Tests

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 3 | A | 9 | Ac | > | Bc | = | n | Pol. | – | $c^4$ | Good | Galt | .7 | Weber |
| | 3 | | 9 | Ac | > | Bc | = | n | | – | | Good | | .7 | + |

## CHART XXVII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

| POINTING | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus None
Vertigo Yes
Past-pointing None
Falling
  Romberg Negative
  Turning head to right No swaying
  Turning head to left No swaying.
  Attempt to overthrow Slight rigidity of pelvic girdle

---

### TURNING

To RIGHT →
  Amp. Good
  Duration 30 Sec.

| To RIGHT | | |
|---|---|---|
| Shoulder from above | 5" to Right | 5" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

To LEFT ←
  Amp. Good
  Duration 32 Sec.

| To LEFT | | |
|---|---|---|
| Shoulder from above | 12" to Left | 12" to Left |

Nystagmus. Normal
Vertigo Normal
Past-pointing Normal

---

### CALORIC

Douche RIGHT ↷
  Amp. Good
  After min. 50 sec.

| Douche RIGHT | | |
|---|---|---|
| Shoulder from above | 4" to Right | 4" to Right.. |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | | |
|---|---|---|
| Head Back Not taken | to | to |
| Amp. | | |

Douche LEFT ↶
  Amp. Good
  After min. 45 sec.

| Douche LEFT | | |
|---|---|---|
| Shoulder from above | 4" to Left | 4" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | | |
|---|---|---|
| Head Back Not taken | to | to |
| Amp. | | |

## CHART XXVIII

Stephen F., 11/18/1914. TESTS OF THE VESTIBULAR APPARATUS Two weeks later

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

Paresis of external rectus.

Shoulder from above

Nystagmus None
Vertigo Yes
Past-pointing None
Falling
Romberg Negative
Turning head to right Not tested
Turning head to left Not tested
Attempt to overthrow Not tested

| POINTING | |
|---|---|
| RIGHT | LEFT |
| Touched | Touched |

### TURNING

To RIGHT →
Amp. Large
Duration 33 Sec.

Limited excursion of left eye to left.

To LEFT ←
Amp. Large
Duration 28 Sec.

To RIGHT
Shoulder from above

Nystagmus Present
Vertigo Normal
Past-pointing Normal

| RIGHT | LEFT |
|---|---|
| 12" to Right | 8" to Right |

To LEFT
Shoulder from above

Nystagmus Present
Vertigo Normal
Past-pointing Normal

| RIGHT | LEFT |
|---|---|
| 12" to Left | 12" to Left |

### CALORIC

Douche RIGHT None
Amp.
After 4 min. sec.

Douche RIGHT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| Touched | Touched |

Nystagmus Absent
Vertigo Absent
Past-pointing Absent
Falling Absent

Head Back →
Amp. Very large

marked vertigo

| RIGHT | LEFT |
|---|---|
| 8" to Right | 6" to Right |

Douche LEFT None
Amp.
After 5 min. sec.

Douche LEFT
Shoulder from above

| RIGHT | LEFT |
|---|---|
| Touched | Touched |

Nystagmus Absent
Vertigo Absent
Past-pointing Absent
Falling Absent

Head Back ←
Amp. Very large

marked vertigo

| RIGHT | LEFT |
|---|---|
| 7" to Left | 9" to Left |

that he was very dizzy and it was noted that a large horizontal nystagmus to the right appeared. The past-pointing was immediately undertaken, and, whereas there had been no past-pointing when he was sitting with the head upright, now there appeared a large past-pointing of each arm in a proper direction—to the left. The right ear was then douched again with the head upright without eliciting any response, and then quickly the head was put backwards. Immediately the normal nystagmus, vertigo and past-pointing appeared.

## COMMENT

This peculiar phenomenon of loss of responses from the vertical semicircular canals of each ear appearing under our own observation within the past two weeks was difficult to explain. We had never observed it before. We were simply confronted with the fact that suddenly the vertical semicircular canals had ceased to react. This failure of reaction could be explained either by a disturbance within the canals themselves or along their pathways. The hearing in this case being normal and the horizontal canals responding normally to stimulation, it was difficult to conceive of a lesion that would pick out only the vertical semicircular canals on each side and leave the rest of the labyrinth intact. It would similarly be difficult to conceive a lesion capable of destroying the vertical semicircular canals fibres within the VIII Nerve and leave the rest of that nerve intact. This disturbance, therefore, must be within the brain-stem between the point of entrance of the VIII Nerve into the brain-stem and the III and IV cranial nuclei. Remembering that the horizontal canals on each side gave perfectly normal responses, this case made it evident that there was a central neuraxial differentiation between the fibres of the horizontal and vertical semicircular canals. The horizontal semicircular canal fibres have been traced histologically to Deiters' nucleus in the medulla oblongata. It appears probable, therefore, that the fibres from the vertical semicircular canals ascend to a higher level and are distributed within the pons. This sudden interference with the function of the vertical semicircular canals fibres of both sides could hardly be attributed to a lesion growing into and involving both sides of the pons. The most probable explanation appears to be an increase of pressure within the IV

## CHART XXIX A

Name Fred L. Age 31 Date 12/10/1915.
Address
Referred by Ear Dispensary, University Hospital, Philadelphia.

DIAGNOSIS:

SUMMARY:

Complains of Headache.

HISTORY:
Dizziness Slight and infrequent
Staggering None
Deafness Impaired hearing, right ear.
Tinnitus No.

NOSE: Negative

THROAT: Negative

EARS:
A. D. Deeply depressed scar over mastoid. Canal stenosed and tender. O.M.C.P.
A. S. Normal

Fistula

Hearing Tests (Dr B. A. Randall.)

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 0 | A | 1/2 | Ac < Bc = n | Pol. | +? | $c^4$ | Fair | Galt | | Weber? |
| = | 2 | | 12 | Ac > Bc = n | | − | | Good | | | |

## CHART XXIX B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT Slight ←
Looking to LEFT Slight →
Looking UP None
Looking DOWN None

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus Negative
Vertigo Negative
Past-pointing Negative
Falling None
Romberg Negative
Turning head to right No swaying.
Turning head to left No swaying.
Attempt to overthrow Not tested.

### TURNING

To RIGHT →
Amp. Fair
Duration 18 Sec.

| | RIGHT | LEFT |
|---|---|---|
| To RIGHT<br>Shoulder from above | Touched | Touched |

Nystagmus Fair
Vertigo markedly impaired. 8 seconds
Past-pointing Absent.

To LEFT ←
Amp. Fair
Duration 14 Sec.

| | RIGHT | LEFT |
|---|---|---|
| To LEFT<br>Shoulder from above | 12" to Left and touched | 24" to Left Touched |

Nystagmus Fair
Vertigo 24 seconds. Normal.
Past-pointing Present but shortened.

### CALORIC

Douche RIGHT
Amp. none
After 4 min. sec.

| | RIGHT | LEFT |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | Touched | Touched |

Nystagmus Absent
Vertigo Absent
Past-pointing Absent
Falling Absent

Head Back →
Amp. Large

Vertigo subnormal

| | RIGHT | LEFT |
|---|---|---|
| Head Back | Touched | Touched |

Douche LEFT ↶
Amp. Barely a twitch.
After 1 min. 34 sec.

Prolonged douching produced practically no nystagmus.

| | RIGHT | LEFT |
|---|---|---|
| Douche LEFT<br>Shoulder from above | Touched | Touched |

Nystagmus Practically absent
Vertigo None
Past-pointing None
Falling None

Head Back ←
Amp. Large.

Vertigo normal

| | RIGHT | LEFT |
|---|---|---|
| Head Back | 5" to Left | 3" to Left. |

ventricle, this pressure interfering with the function of the vertical canals fibres in its floor.

A bilateral subtentorial decompression was performed on November 19, 1914, and the surgeon's report reads: "The cortex of both cerebellar hemispheres appeared to be normal, except perhaps that the meninges appear a trifle injected. There was, however, no sign of a tubercular deposit nor of any tubercles either on the cerebellar hemispheres or on the meninges. There was no evidence of tumor."

Autopsy was refused, but the clinical picture was one of undoubted meningitis.

Case 15.—Mr. Fred L., age 30. Presented himself to the ear dispensary of the Hospital of the University of Pennsylvania on December 10, 1915; he had been suffering with severe headaches and wanted to ascertain whether some previous ear operations that he had had were in any way responsible for the headaches. His right ear has been operated on seven times -the last time twelve years ago. Following this last operation he had no trouble until two weeks ago, when he had a convulsion, which came on so suddenly that he had no recollection of it. He was taken to the Medico-Chirurgical Hospital in an ambulance in an unconscious condition. The following day he recovered consciousness, but had another convulsion two days later while in the hospital. Four days later, however, he felt perfectly well and insisted on leaving the hospital. He remained well except for an occasional pain in the head, which, however, was very severe while it lasted.

When he appeared at the dispensary he presented a picture of a normal, healthy man. The accompanying chart (XXIX) gives the data of the ear examination at this time. We note that the right or operated ear shows impaired hearing; this deafness however, is of the obstructive type and the cochlea is apparently functionating. The hearing in the left ear is normal. Turning to the right shows a marked contrast between the nystagmus and the past-pointing. The caloric test shows an absence of responses on stimulation of the vertical canals of both ears. The right horizontal canal shows a normal nystagmus, but an absence of past-pointing. The left horizontal canal produces normal responses.

These impaired or absent responses evidently indicated an organic lesion of some sort. Further, the ear examination suggested a central lesion because the cochleas were normal and each horizontal semicircular canal gave at least one normal response. The absence of responses from the vertical semicircular canals of both ears could not be accounted for by a lesion either within the labyrinths or in the VIII Nerves, but indicated an interruption along their intracranial pathways within the pons. The impaired vertigo from the right horizontal canal probably indicated a disturbance in the region of the right inferior cerebellar peduncle. The report of the ear examination was as follows: "There is evidence of pressure within the IV ventricle. In view of the history of so many ear operations, these findings would indicate that the patient is in a serious condition." This report was made with some misgiving, because of the apparent good health of the patient. The patient was advised to enter the hospital; this advice was not followed and the patient went home. Six days after this examination the patient had another convulsion associated with unconsciousness and was rushed to the University Hospital in the ambulance. The following day he regained consciousness and appeared normal again, except that he complained of an agonizing headache. The patient's grip was very good and equal in both hands. There was no loss of power in either lower extremity. Sensation to touch and pain was normal throughout the entire body. Pupils were equal and responded normally to light, accommodation and convergence—in fact, careful neurologic examination failed to reveal any signs of intracranial mischief.

The right ear—the one that had had so many operations—was again operated on by Dr. B. Alexander Randall and nothing definite was discovered beyond diminished pulsation of the dura. There was no evidence of extra-dural or intra-dural abscess. Following this operation the patient was very restless and entirely unmanageable. It was recalled that at the time of the operation twelve years ago when this patient was in the same hospital and operated on also by Doctor Randall, he was equally unmanageable. This appeared to strengthen the general consensus of opinion that they were dealing with a functional case. It was difficult to conceive of anything except hysteria which could account for these sudden and prolonged attacks of unconsciousness with convulsions, from which the patient would emerge apparently well the following day. Lumbar puncture had proven negative, but two days after the operation when it was repeated, 30 c.c. of a turbid cerebrospinal fluid was withdrawn. Microscopic examination of this fluid revealed an intracellular diplococcus. There also occurred a rise of temperature with leucocytosis. Very suddenly there appeared a spontaneous vertical nystagmus downward, associated with an occasional horizontal nystagmus to the right. The patient died on the thirteenth day after admission to the hospital. The post-mortem examination revealed a large collection of pus filling the IV ventricle, which had apparently reached there through the aqueduct of Sylvius from the III and lateral ventricles, which were filled with pus from a ruptured abscess in the right tempero-sphenoidal lobe. This abscess had communicated with the tegmen by a tiny pin-hole opening for a distance of about half an inch.

Case 16.—Edgar S., age 16. The patient was admitted to the Medico-Chirurgical Hospital November 5, 1914, with the following history: He was apparently in perfect health until three months ago, when he began to complain of morning headaches. Coincident with the headache there appeared occasional double vision. His sight began to fail and for the past three weeks he has been practically blind.

The following are the notes of the examination made at the hospital: "The patient stands with feet wide apart. Station somewhat uncertain, with a tendency to fall to the left. Gait is fair, with a tendency to walk to the left. Pupils are widely dilated. No demonstrable paralysis of the extraocular muscles. Opens and closes eyes equally well. No weakness in the distribution of the facial nerves. No weakness of the muscles of mastication. No loss of power of the upper or lower extremities. Knee jerks absent. Achilles jerks present. In attempting to touch

## CHART XXX A

Name Edgar S. Age 16 Date 11/27/1914.
Address
Referred by Dr Charles Potts.

DIAGNOSIS:

SUMMARY:

Complains of Blindness, staggering and occasional vertigo.

HISTORY:
Dizziness Present only when he shakes his head quickly. Is associated with nausea.
Staggering Yes
Deafness No
Tinnitus No

NOSE: Septum deviated to Right, high up. Spur low down along floor on Left side.

THROAT: Tonsils large.

EARS:
A. D. Drum head freely movable – normal.
A. S. Drum head slightly retracted but freely movable.

Fistula Negative

Hearing Tests

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| A | 2 | A | 4 | Ac = Bc = n | Pol. | | $c^4$ | Good | Galt | Weber to Right. |
| = | 1½ | | 4 | Ac > Bc = n | | | | Good | | |

## CHART XXX B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT ←
Looking to LEFT →
Looking UP None
Looking DOWN None

| POINTING | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | 1" to Right<br>Touched |

Nystagmus Present, larger on looking to Left
Vertigo None
Past-pointing None
Falling None
Romberg Some swaying.
Turning head to right does not increase swaying.
Turning head to left " " " "
Attempt to overthrow not tested.

---

### TURNING

To RIGHT →
Amp. Large
Duration 30 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 5" to Right<br>Touched | 3" to Right<br>3" - Right |

Nystagmus Exaggerated
Vertigo Impaired
Past-pointing Shortened

To LEFT ←
Amp. Large
Duration 35 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 4" to Left | Touched |

Nystagmus Exaggerated
Vertigo Impaired
Past-pointing Absent of Left arm to Left.

---

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 30 sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 2" to Right<br>Touched | Touched |

Nystagmus Normal
Vertigo Subnormal
Past-pointing Almost absent
Falling Almost absent.

| | | |
|---|---|---|
| Head Back Not tested | to | to |

Amp.

Douche LEFT ↶
Amp. Large, slow.
After 1 min. sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 3" to Left | Touched<br>2" to Right. |

Nystagmus Normal
Vertigo Subnormal
Past-pointing Absent Left arm to Left
Falling Absent.

| | | |
|---|---|---|
| Head Back Not tested | to | to |

Amp.

the end of the nose with the left index finger there is a tendency to point to the left. There is adiadokokinesis of the left arm. In attempting to put the left heel on the right knee he touches a point above the knee. Flexing and extending the left thigh on the body is somewhat more jerky than on the right. Movements of the trunk are well performed."

Subsequent examination showed an improvement in these findings, and the following notes were dictated: "Finger-to-nose test seems to be better performed by the left hand than at the previous examination. The rotation of the left arm is better performed than at first—less marked adiadokokinesis. Heel-to-knee test well performed on both sides. Straightening the lower extremities after the knees have been flexed on the body appears to be well performed."

*Eye examination by Dr. L. Webster Fox* showed choroid involved, maculæ and retinæ edematous. Vision, only shadow and light. Field of vision, nil. Color perception, nil.

X-ray examination on November 27, 1914. Diagnosis: Pituitary tumor. Partial absorption of bone salts in the left cerebellar region. The sella turcica is enlarged and the posterior clinoid process is elongated and partially absorbed. There is an area of absorption of lime salts about ¾ of an inch in diameter bordering on the left side of the foramen magnum.

The Wassermann test of the blood was strongly positive.

Examination of the vestibular apparatus was made on November 27, 1914, and findings recorded on the accompanying chart (XXX). The exaggerated nystagmus after turning is indicative of an irritation of the brain-stem, as from pressure. Turning and douching both produced a good nystagmus, from the horizontal canals and the vertical canals of both ears; this would indicate that the medulla oblongata and pons themselves were intact. There is a striking absence of past-pointing of the left arm to the left, both after turning to the left and after douching the left ear with cold water—both of which should normally produce a past-pointing of the left arm to the left. The following report was made: "The ear tests suggest a cerebellar lesion, in the left lateral hemisphere, in the region governing the outward pointing centre of the left upper extremity. The following appear normal: (1) Both labyrinths, (2) Both VIII Nerves, (3) Medulla oblongata, (4) Pons."

Operation: Bilateral cerebellar exposure, dura found to be tense and on opening the dura on a wide exposure no removable tumor mass was found.

*Autopsy.*—The following is the report of Dr. T. H. Weisenburg: "There is a softening of the left cerebellar hemisphere, taking in chiefly the anterior internal portion of the quadrangular lobe, extending as a gelatinous mass in the left cerebellopontile angle and contiguous with the medulla oblongata. The softening extends into the left side of the vermis and also involves the right dentate nucleus. The aqueduct of Sylvius is almost obliterated by the tumor mass. The right cerebellar hemisphere and the right side of the vermis are normal. The outer portion of the left quadrangular lobe is also normal."

Case 17.—Agatha I., age two and one-half.

This case illustrates the value of a thorough testing of the vestibular apparatus in cases of intracranial complication in the course of middle-ear suppuration. Three weeks prior to this ear examination a simple mastoid operation had been done on the right ear. Three days prior to the examination, during what appeared to be an uninterrupted convalescence, the patient began to be semi-stuporous and the pulse became slower and slower. There were no chills and the temperature remained about normal. It was evident that there was some intracranial complication. The differential diagnosis lay between lateral sinus or jugular thrombosis, meningitis, cerebellar abscess, or tempero-sphenoidal abscess.

The ear examination on January 7, 1916, showed the following:

(1) Douching the right ear on two different occasions failed to produce any response whatsoever. The ear was douched both with the head in the upright position, stimulating the vertical canals, as well as with the head 60° backward, stimulating the horizontal canal. Neither test elicited any nystagmus.

(2) Douching the left ear with cold water produced a conjugate deviation to the left, after one minute and thirty seconds. On two occasions the left ear was douched with hot water, producing a conjugate deviation of both eyes to the right.

There was an occasional spontaneous vertical nystagmus upward.

The clinical picture of itself was sufficient to exclude lateral sinus or jugular thrombosis as well as meningitis. The presence of a slow pulse was most suggestive of a purulent collection within the cranium. There was nothing, however, which would indicate whether the pus was in the cerebellum or in the tempero-sphenoidal lobe. The responses to the ear stimulation, however, were very suggestive. A temporo-sphenoidal abscess could hardly account for a total absence of reactions from the right ear as well as the occasional spontaneous vertical nystagmus. This total absence of responses from the right ear clearly indicated a destruction of either the labyrinth, the VIII Nerve, or a collection of pus in the right cerebello-pontile angle involving the VIII Nerve. Since the clinical picture pointed to an intracranial abscess the question of a purulent labyrinthitis could be disregarded and it seemed most probable that the case was one with an abscess in the cerebellum in its anterior portion pressing into the angle or a collection of pus within the angle itself. At any rate the surgical indications were an exploration of the *posterior* and not the middle fossa of the skull. On the strength of this information the right cerebellum was explored and three teaspoonfuls of fetid pus evacuated.

Case 18.—Mr. Harold S., age 16. This patient was examined by us at a clinic given by Dr. G. E. Shambaugh in Chicago June 17, 1915. The examination of the vestibular apparatus was carried out without any knowledge of his previous history, and the report was made exclusively on the basis of the findings obtained by turning and douching. The accompanying chart (XXXI) presents the data obtained at this examination. A glance at the markedly abnormal responses shown on the vestibular chart indicates at once an organic lesion in some portion of the vestibular mechanism. The labyrinths and VIII Nerves can be excluded because the hearing is normal and turning in both directions produced a large nystagmus. The lesion, therefore, is central. The most striking abnormalities are the subnormal vertigo and the peculiar past-pointing. Nausea, dizziness and vomiting were strikingly absent in spite of the fact that the entire series of tests was carried out at one sitting

## CHART XXXI A

Name Harold S. Age 16 Date 6/17/1915
Address
Referred by Dr Peter Bassoe

DIAGNOSIS:

SUMMARY:

Complains of
HISTORY:
Dizziness
Staggering
Deafness
Tinnitus
} Not supplied

NOSE:
THROAT:
} Not examined

EARS:
A. D. Freely movable, Intact.
A. S. Freely movable, Intact.

Fistula Not tested

Hearing Tests Dr Shambaugh and Dr Brunn reported normal hearing

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ \| | A \| | Ac | Bc | n | Pol. \| | $c^4$ \| | Galt \| |
| | | Ac | Bc | n | | | |

## CHART XXXI B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT Faint ←
Looking to LEFT None
Looking UP None
Looking DOWN None

Shoulder from above

Nystagmus None
Vertigo None
Past-pointing None
Falling None
Romberg Not-tested
Turning head to right Not-tested
Turning head to left Not tested
Attempt to overthrow Not tested

| POINTING | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

---

### TURNING

To RIGHT →
Amp. Large
Duration 44 Sec.
Irregular

To RIGHT
Shoulder from above

Nystagmus Exaggerated
Vertigo Subnormal
Past-pointing Impaired and perverted

To LEFT ←
Amp. Large
Duration 40 Sec.
Large amplitude persisted to the end.

To LEFT
Shoulder from above

Nystagmus Exaggerated
Vertigo Subnormal
Past-pointing Impaired

| POINTING | RIGHT | LEFT |
|---|---|---|
| To RIGHT, Shoulder from above | 3" to Left | 4" to Right |
| To LEFT, Shoulder from above | 4" to Left | 2" to Left and touched |

---

### CALORIC

Douche RIGHT ↶ to Right
Amp. Good
After 2 min. 4 sec.

Douche RIGHT
Shoulder from above

Nystagmus Inverse
Vertigo Absent
Past-pointing Almost absent
Falling Absent

Head Back Not tested
Amp.

Douche LEFT ←
Amp. Fair
After min. 38 sec.

Douche LEFT
Shoulder from above

Nystagmus Perverted
Vertigo Almost absent
Past-pointing Almost absent
Falling Absent

Head Back Not-tested
Amp.

| POINTING | RIGHT | LEFT |
|---|---|---|
| Douche RIGHT, Shoulder from above | Touched | 3" to Right |
| Head Back | to | to |
| Douche LEFT, Shoulder from above | 2" to Left | 1" to Right. |
| Head Back | to | to |

and in rapid succession. This absence of the normal vertigo and past-pointing naturally at once suggests a cerebellar lesion. The medulla oblongata would appear to be uninvolved, because stimulation of both horizontal semicircular canals, by turning both to the right and to the left, produced a large nystagmus; the prolonged duration and large amplitude of this nystagmus, however, would suggest irritation by pressure. Stimulation of the right vertical semicircular canals produced the remarkable phenomenon of an *inverse* nystagmus; stimulation of the left vertical semicircular canals produced a *perverted* nystagmus. These would suggest an involvement of the brain-stem by infiltration or pressure from without. The following report was made: "The ear examination suggests a lesion of both sides of the cerebellum, chiefly the right, with pressure against the brain-stem."

Following this statement Dr. Peter Bassoe then gave the full history of the case as follows: "Headache began to appear in November, 1914, but not very severe until two weeks ago. Morning vomiting began six weeks ago, and tendency to staggering has been noticed since January, with tendency to fall backward and to the right. For three weeks blurring when reading and some degree of lateral diplopia. Vertigo is sometimes complained of. The headache was most severe in the forehead and vertex. He attended school until March 29." Dr. E. V. L. Brown discovered bilateral papillitis on April 1st, whereas only a few weeks before another ophthalmologist had found the eye grounds normal.

*Examination.*—Pupils react normally; the left slightly wider than the right; lateral nystagmus on looking to either side, more so when looking to the right. Slight lateral diplopia; fields roughly normal; bilateral choked disc. The patient can read fine print with either eye. Sense of smell normal; taste apparently a little sluggish. Watch heard at three feet left ear; one foot right ear. Tongue protruded slightly to the left. In smiling, however, the mouth is drawn more to the left. The forehead wrinkles equally; shutting of eyes apparently stronger on right side. Muscles of mastication of normal strength. Right corneal reflex weaker than left; sensation everywhere normal. Moderate ataxia of right hand, very little of left hand. Slight adiadokokinesis of right forearm. Wrist jerk not obtained; elbow jerks weak, especially left; abdominal reflexes weak; cremasteic present, weak; knee and ankle jerks not obtained; plantar reflex normal on both sides.

*Diagnosis.*—Tumor of posterior fossa on right side, probably in cerebellum or cerebello-pontile angle. Operation advised.

The operation was performed in two stages by Dr. Dean D. Lewis. The two operations were eight or ten days apart. At the second one, performed on April 29th, a cyst was discovered deep in the right lateral lobe of the cerebellum and about 25 c.c. of straw-colored fluid evacuated. When the dura first was opened and the cerebellum palpated no difference in tension between the two halves could be made out, nor did inspection offer any clue to the site of the lesion. The cyst was discovered by the introduction of an aspirating needle.

The patient improved very rapidly.

It is of interest to note that the ear-examination was made after the patient had recovered from this operation; Doctor Bassoe and Dr. Dean Lewis knew the location of the lesion and it was therefore a good opportunity to test the accuracy of an examination of the vestibular apparatus.

Case 19.—Mr. Leo S., age 20. Patient admitted to the Hospital of the University of Pennsylvania on May 10, 1916, complaining of vertigo, loss of memory and headache. Seven months prior to admission it was noted by the patient and his friends that his violin playing had perceptibly deteriorated. It was noted at the same time that his general mentality was failing. Five months ago he began to complain of dizziness; objects appeared to be moving from right to left. For the last two months he has had a staggering gait. Headache appeared two weeks ago and is persistent; it is mostly occipital, but frequently also frontal. For the last four months his vision has been failing. Gives no history of having had any convulsions, paralysis or objective sensory disturbances.

The previous medical and family history are negative.

*Physical examination.*—Frequent tremor of neck muscles with nodding movements of the head. Action of facial, pharyngeal and tongue muscles unimpaired. Biceps reflexes prompt and equal. Knee jerks equal, but somewhat more sluggish than the other reflexes. Achilles prompt and equal. Plantar stimulation of either foot induces plantar flexion. No ankle clonus. Tactile and pain sensation are normal throughout. Grip strong and equal. Patient unsteady. Swaying is increased when eyes are shut. Gait tends to ataxia and inco-ordination. Movements of hands are clumsy. Adiadokokinesis in both hands. Finger-to-nose test and heel-to-knee test are done with moderate accuracy. Sense of position and movement as well as steriognostic sense are normal. In bending backward does not flex knee; no cerebellar catatonia. There is no asynergy in flexion of lower limbs.

*Eye Examination.*—Vision right eye, 6/12ths; left, 6/16ths. There is noted a definite striation of the upper margins of both nerves. Fields normal. There is a paresis of the internal and external rectus of the left eye.

X-ray examination by Dr. Pancoast negative.

Lumbar puncture shows the spinal fluid under a pressure of 8 m.m.; it is clear; contains very few cells.

Wassermann is negative. Globulins slightly positive.

The results of the examination of the vestibular apparatus are recorded on the accompanying chart (XXXII). It will be noted that the patient is unable to look to the right with the left eye. Ear stimulation, however, does cause a definite contraction of the internal rectus of the left eye; this movement is not complete and does not draw the eye completely inward, but it shows definitely that the left III nucleus is not completely paralyzed (unless certain fibres come from the right III nucleus to the left internal muscle).

The marked vertical nystagmus upward and also vertical nystagmus downward indicate a lesion of the brain-stem either by infiltration or pressure. After ear stimulation from every canal except the left horizontal, there is a *perverted* nystagmus; on turning both to the right and to the left there is produced not a pure

## CHART XXXII A

Name Leo S. Age 20 Date May 10, 1916.
Address
Referred by University Hospital.

DIAGNOSIS:

SUMMARY:

Complains of Dizziness, staggering, headaches.

HISTORY:
Dizziness Since September following operation for appendicitis.
Staggering Yes
Deafness 2 months duration, Right ear worse than Left.
Tinnitus No.

NOSE: Not examined

THROAT: Not examined

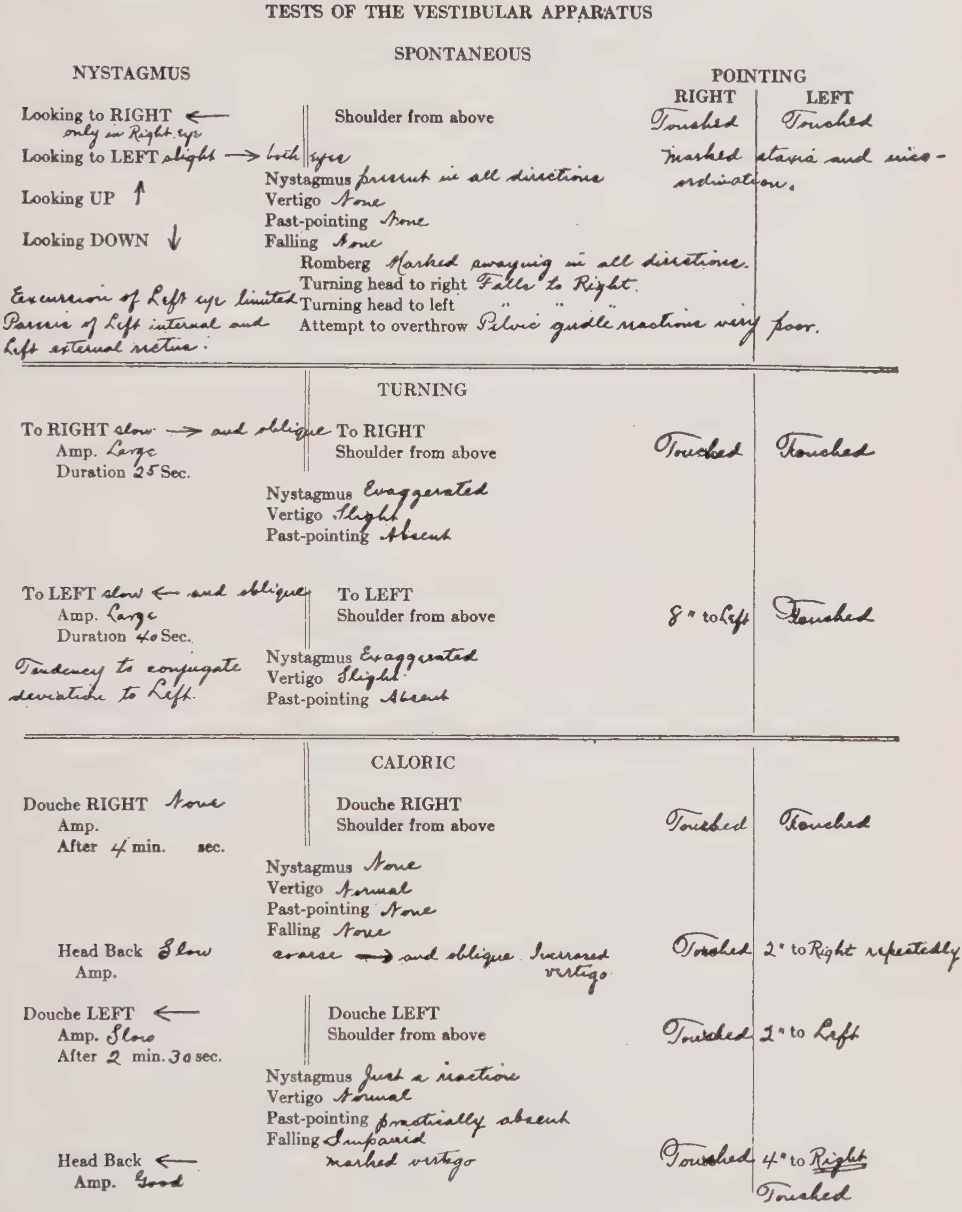
EARS:
A. D. Same as left.
A. S. Small amount of cerumen. M.T. atrophic; hammer handle bound down

Fistula

Hearing Tests

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 1 | A | 10 | Ac > Bc < n | Pol. | | $c^4$ | Good considerably | Galt | .6 |
| | | | 6 | Ac > Bc < n | | | | Good slightly. | | .6 |

## CHART XXXII B



### TESTS OF THE VESTIBULAR APPARATUS

#### SPONTANEOUS

| NYSTAGMUS | | POINTING RIGHT | POINTING LEFT |
|---|---|---|---|
| Looking to RIGHT ← only in Right eye | Shoulder from above | Touched | Touched |
| Looking to LEFT slight → both eyes | | marked ataxia and incoordination. | |
| Looking UP ↑ | Nystagmus present in all directions<br>Vertigo None<br>Past-pointing None<br>Falling None | | |
| Looking DOWN ↓ | Romberg Marked swaying in all directions.<br>Turning head to right Falls to Right.<br>Turning head to left " " "<br>Attempt to overthrow Pelvic girdle reactions very poor. | | |
| Excursion of Left eye limited. Paresis of Left internal and Left external rectus. | | | |

#### TURNING

| | | | |
|---|---|---|---|
| To RIGHT slow → and oblique<br>Amp. Large<br>Duration 25 Sec. | To RIGHT<br>Shoulder from above | Touched | Touched |
| | Nystagmus Exaggerated<br>Vertigo Slight<br>Past-pointing Absent | | |
| To LEFT slow ← and oblique<br>Amp. Large<br>Duration 40 Sec. | To LEFT<br>Shoulder from above | 8" to Left | Touched |
| Tendency to conjugate deviation to Left. | Nystagmus Exaggerated<br>Vertigo Slight<br>Past-pointing Absent | | |

#### CALORIC

| | | | |
|---|---|---|---|
| Douche RIGHT None<br>Amp.<br>After 4 min. sec. | Douche RIGHT<br>Shoulder from above | Touched | Touched |
| | Nystagmus None<br>Vertigo Normal<br>Past-pointing None<br>Falling None | | |
| Head Back Slow<br>Amp. | coarse → and oblique. Increased vertigo | Touched | 2" to Right repeatedly |
| Douche LEFT ←<br>Amp. Slow<br>After 2 min. 30 sec. | Douche LEFT<br>Shoulder from above | Touched | 2" to Left |
| | Nystagmus Just a reaction<br>Vertigo Normal<br>Past-pointing practically absent<br>Falling Impaired<br>marked vertigo | | |
| Head Back ←<br>Amp. Good | | Touched | 4" to Right<br>Touched |

horizontal nystagmus, but a mixed horizontal and oblique, as well as an occasional upward. This further suggests brain-stem involvement.

After turning there is a noticeably long duration of the nystagmus. This usually indicates a neuraxial disturbance and of itself points to pressure rather than destruction of the fibres.

After turning, the nystagmus is large and very slow; this suggests an impairment of the cerebral fibres which fail to bring about a quick return movement of the eye.

Stimulation of the right vertical canals fails to produce any nystagmus, indicating a lesion in the upper part of the pons. Inasmuch, however, as the vertical canals of the right did produce vertigo, the lesion indicated would be in the region of the posterior longitudinal bundles rather than of the right middle cerebellar peduncles. Vertigo was also produced by both horizontal canals; this suggests uninvolved inferior cerebellar peduncles.

All of the semicircular canals produced vertigo, although it was impaired from some of them. This suggests that the decussation of the superior cerebellar peduncles in the neuraxis at the base of the cerebral crura is not involved. This is rather surprising, because the impairment of the quick component of nystagmus suggests a lesion very near this point.

The fibres from the left vertical canals in the upper portion of the pons appear definitely encroached upon, in the region of the posterior longitudinal bundles, but not completely blocked as are the right vertical canals fibres.

The horizontal canals of both sides cause more vertigo than the vertical canals; this bears out other data suggesting that the lesion is in the upper part of the cerebellum and brain-stem rather than in the lower part.

Pointing. Spontaneous pointing shows a slight ataxia of both arms. The right arm occasionally points towards the left, this *per se,* would suggest a lesion of the outward pointing centre of the right lateral cerebellar hemisphere. This is further borne out by the inability to cause the right arm to point outward by ear-stimulation. There is a definite absence of past-pointing outward of the right arm after turning to the right, after douching the right ear with cold water, or after douching the left ear with hot water; all of these tests should cause the right arm to point outward. It is true that after turning, the left arm also fails to point to the right, but cold water to the right horizontal canal produces a definite past-pointing of 4 inches of the left arm to the right, and douching the left horizontal canal with hot water also causes a correct past-pointing of the left arm to the right. All this points to a lesion of the outward pointing centre of the right lateral cerebellar hemisphere; this centre is supposed to be located on the upper surface of the cerebellum toward the median line in the quadrangular lobe. (Barany's idea, however, is that this centre is in the semilunar lobe, both superior and inferior, in the region of the large fissure.)

Inasmuch as the patient experiences vertigo from ear-stimulation, and yet shows marked absence of past-pointing of both arms, in both directions, more particularly the right arm, it is suggested that the *motor* fibres from the cerebral cortex are interrupted, either in the brain-stem or in the cerebellum itself.

The pelvic girdle reactions are so markedly impaired that a lesion of the vermis is indicated.

There is evidence, therefore, of involvement of the right lateral cerebellar hemisphere, the vermis, the left lateral hemisphere of the cerebellum, the upper posterior portion of the pons, and the posterior portion of the junction of the cerebral crura. It is evident, therefore, that some of these phenomena are due to pressure. However, as there is such marked involvement of the brain-stem itself, it would appear that the lesion, if a tumor, is inoperable. The most flagrant absence of reaction is in the outward pointing of the right arm. The best explanation would be an infiltrating glioma involving the outward pointing centre of the right lateral cerebellar hemisphere, extending over to the vermis and entering the pons at its upper half, and extending into the base of the junction of the cerebral crura.

The patient was operated upon on June 21, 1916, by Dr. C. H. Frazier. Report of operation: "A suboccipital craniectomy. Wide exposure of both cerebellar hemispheres. Superior, inferior and lateral aspects of the hemispheres seem to be entirely free from suspicion of tumor. In the median axis, however, coming up from the two hemispheres, was seen what appeared to be a gliomatous growth of infiltrating character, soft and gray in appearance, a portion of which was removed for examination. Possibility of involvement of both hemispheres and the pons as indicated in the ear report seems quite probable."

CASE 20.—Richard Z., age 13. Patient was admitted to the Jefferson Hospital on April 19, 1917, complaining of inability to walk, blindness and general weakness.

About one year ago, while sleighing, patient was thrown to the ground; he was dazed by the fall, but soon after being carried into the school room he became normal again. In April, 1916, that is, about a month later, he began to have severe headaches. A month later he began to vomit. This vomiting recurred with greater or lesser frequency for several months. Eight months ago the patient had a "fainting spell" followed by convulsive movements of the hands and legs; the head was drawn back and there was a rolling of the eyes. Similar convulsions occurred rather frequently until about two weeks before admission to the hospital. Patient has been unable to walk and was confined to bed during the five months immediately preceeding his admission to the hospital. Two months ago patient became dull and the speech became explosive.

Family and previous medical history are negative.

*Physical Examination.*—Patient emaciated and unable to walk. Knee jerks absent. Double Babinski. No loss of sensation. General weakness but no paralysis of extremities. Finger-to-nose test shows a loss of co-ordination. There is adiadokokinesis of the right side.

*Eye Examination.*—No light perception in either eye. Pupils do not react to light, but do react to convergence and accommodation. Ocular movements unimpaired. There is a choking of the disc in both eyes—2 diopters in the right and 5 diopters in the left. There is also evidence of a secondary atrophy in both eyes.

Wassermann negative.

Findings of the examination of the vestibular apparatus are recorded on the accompanying chart (XXXIII).

## CHART XXXIII A

Name Richard J. Age 13 Date April 18, 1917.
Address
Referred by Jefferson Hospital

DIAGNOSIS:

SUMMARY:

Complains of Inability to walk, headache, blindness

HISTORY:
Dizziness Yes
Staggering Yes
Deafness None
Tinnitus Yes

NOSE: Not examined

THROAT: Not examined

EARS:
A. D. Drum head normal
A. S. " " "

Fistula Not tested

Hearing Tests

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| A | A | Ac | Bc | n | Pol. | $c^4$ | Galt |
| = | | Ac | Bc | n | | | |

Hearing normal, both ears.

## CHART XXXIII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | 1" to Right |
| | Incoordination. | |

Nystagmus None
Vertigo None
Past-pointing practically none
Falling
Romberg
Turning head to right
Turning head to left
Attempt to overthrow
} Unable to stand up.

### TURNING

To RIGHT →
Amp. Large
Duration 28 Sec.

Conjugate deviation to the Right but at first nystagmus to Left.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| To RIGHT<br>Shoulder from above | Past-pointing correct but shortened | Past-pointing correct to but shortened. |

Nystagmus Normal
Vertigo apparently subnormal
Past-pointing Impairment

To LEFT ←
Amp. Large
Duration 30 Sec.

At first horizontal to Right then conjugate deviation to Left.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| To LEFT<br>Shoulder from above | Past-pointing correct but shortened | Past-pointing correct but shortened. |

Nystagmus Exaggerated
Vertigo subnormal
Past-pointing Impaired

### CALORIC

Douche RIGHT → ↷
Amp. Fair
After 1 min. 20 sec.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | Touched | 3" to Right. |
| Head Back | Touched | Touched |

Nystagmus Perverted
Vertigo Subnormal
Past-pointing absent Right arm
Falling Subnormal

Head Back Conjugate deviation to Right
Amp.

Douche LEFT mixed nystagmus
Amp.
After 1 min. 30 sec.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Douche LEFT<br>Shoulder from above | 8" to Left | 7" to Left |
| Head Back | 2" to Left | 3" to Left. |

Nystagmus Perverted
Vertigo Subnormal
Past-pointing Normal
Falling Normal

Head Back Conjugate deviation to Left
Amp.

It will be noted that we are obviously dealing with a central lesion. The following are the most striking results of the ear-examination:

(1) Impaired vertigo from the stimulation of all of the semicircular canals.

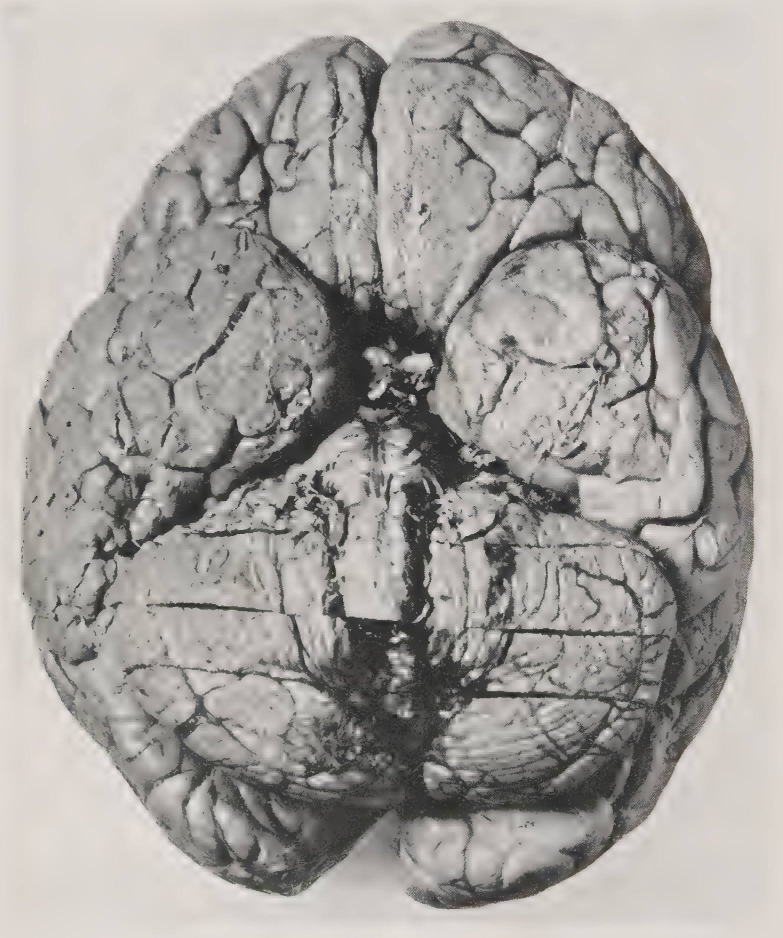


FIG. 118.—Cerebellar cortex uninvolved. (Case 20).

(2) A perverted nystagmus obtained on stimulation of the vertical semicircular canals of both sides.

(3) A marked tendency to a conjugate deviation from the stimulation of both horizontal semicircular canals. This indicates that the cerebral component of the nystagmus to right and to left is impaired. The impairment of vertigo from the

stimulation of both ears would suggest a lesion at the point where all the vertigo fibres come together, namely, at the decussation of the two superior cerebellar peduncles. This is further corroborated by the interference of both cerebral components of nystagmus. Patient's inability to sit up or stand up was due to incoordination of his trunkal muscles. This would suggest an involvement of the vermis of the cerebellum. The poor responses in past-pointing would also suggest an involvement of the cerebellum. Both arms, however, did past-point in the correct direction, although only slightly. This would indicate that the cerebellar *cortex* itself is practically intact. The following report was made: "The lesion is probably located at the junction of the anterior superior portion of the cerebellum with the brain-stem. The lesion appears to spring from the brain-stem itself at the region of the posterior corpora quadrigemina, or from the posterior aspect of the junction of the cerebral crura just above the region of the III nuclei and is centrally located.

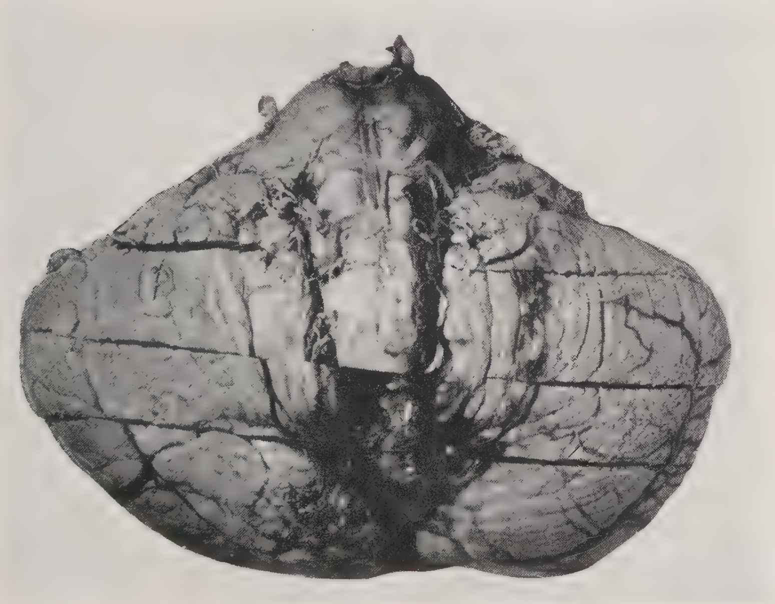


FIG. 119.—Cerebellar cortex intact. (Case 20).

" The cerebellar phenomena would appear to be due to an involvement of the motor fibres on the way to the cerebellum, rather than a lesion of the cerebellar cortex itself."

*Autopsy* by Doctor Funk.—Upon removing the skull cap a considerable amount of clear, colorless fluid escapes, apparently coming from beneath the membranes in region of cerebellum. Transverse sections through both cerebral hemispheres reveal nothing abnormal. There is, however, an opening in the cerebellum which leads into the brain substance for a distance of 6.5 c.m. The walls of the collapsed opening are smooth, but the dura bordering it has rough and ragged edges. Upon slight pressure a small amount of fluid, similar to that which escapes when the skull cap was removed, can be seen oozing from the opening (Figs. 118, 119, 120).

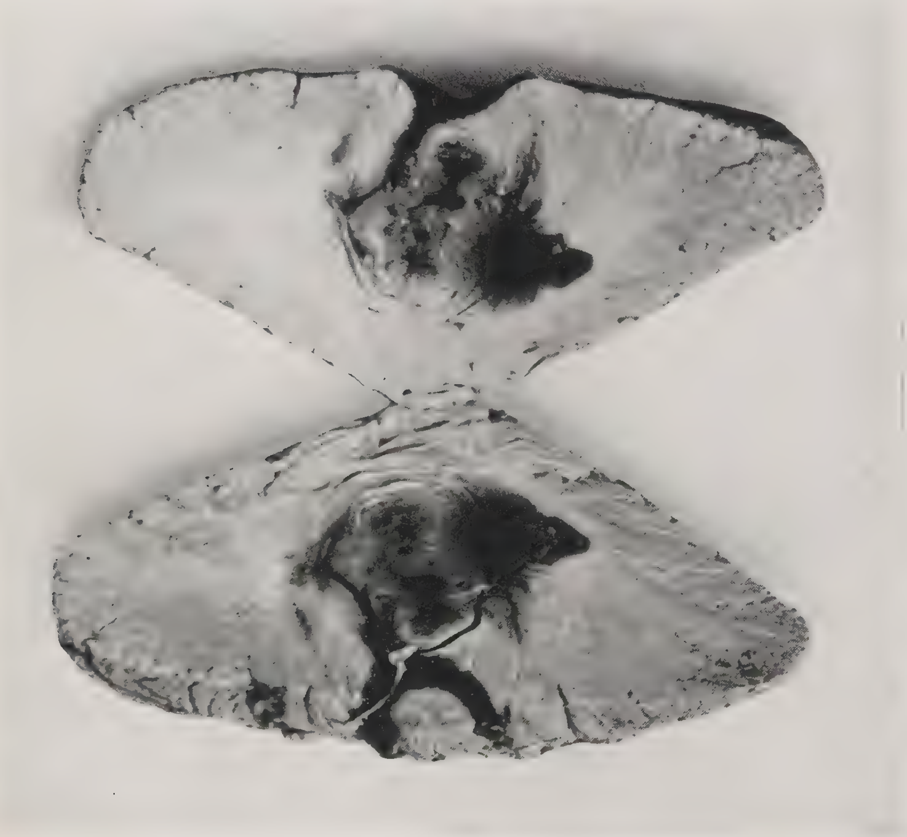


Fig. 120.—Cerebellar cortex intact. Cyst involving vermis. (Case 20).

Case 21.—Miss C., age 23. The patient was apparently in perfect health one month before her death. Her illness began with intense and frequently recurring pain in the head, attacks of nausea and at times projectile vomiting. Dr. Charles K. Mills made the following notes of his examination: "Complete atonia or flaccid paralysis of the left upper and lower extremities; some paresis of the face of cerebral type; Jacksonian epilepsy, beginning in the left side of the face; asteriognosis; some impairment of cutaneous and muscular sensibility; somewhat increased deep and superficial reflexes; negative Babinski; considerable mental hebetude."

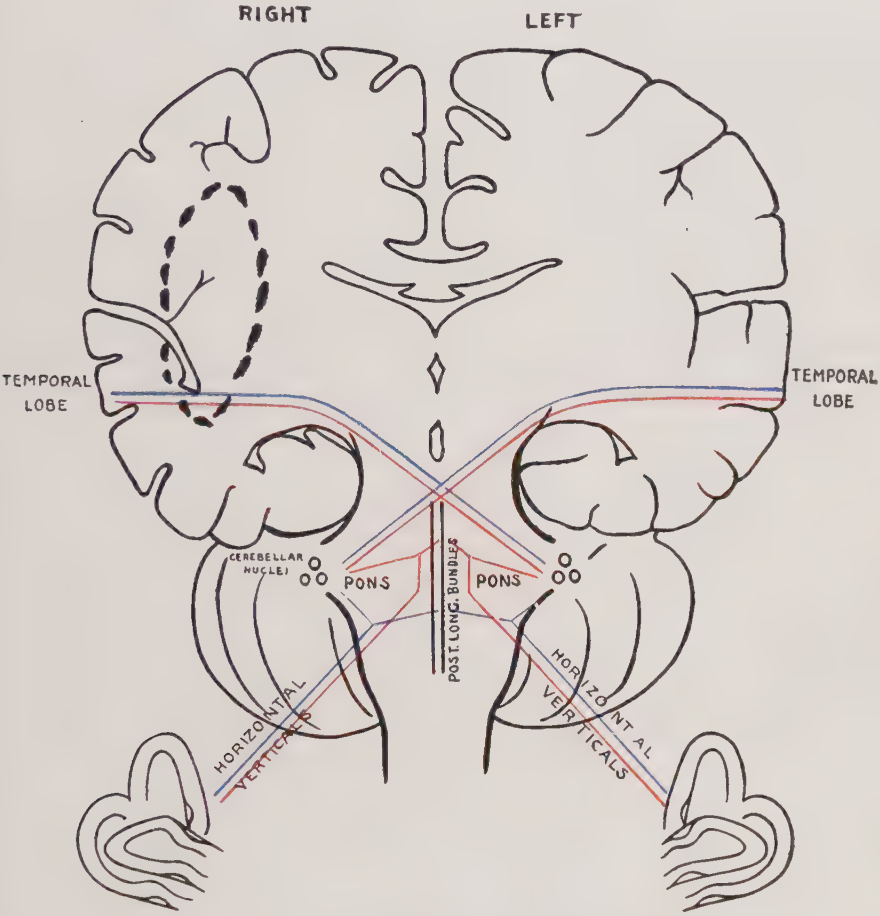


Fig. 121.—Abscess, sub-cortical, right parietal region.

Dr. Holloway reported marked papillœdema. Diagnosis by Dr. Mills: "Tumor involving both the parietal and frontal regions on the right side."

On the accompanying chart (XXXIV) we note the details of the ear examination. There is no spontaneous nystagmus and no spontaneous past-pointing of the right arm; the pointing tests of the left arm can not be undertaken because of the

## CHART XXXIV A

Name Miss C. Age 23 Date Nov. 6, 1916.
Address
Referred by Dr Chas. K. Mills

DIAGNOSIS:

SUMMARY:

Complains of

HISTORY:
Dizziness Yes
Staggering Yes
Deafness No
Tinnitus No.

NOSE:

THROAT:

EARS: A. D. Slightly impaired mobility – otherwise normal.
A. S. Same

Fistula

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 3 | A | 12 | Ac > Bc > n | Pol. | | $c^4$ | Good | Galt | .7 | Weber |
| | 5 | | 10 | Ac > Bc > n | | | | Good | | .7 | + |

## CHART XXXIV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | Paralyzed |

Nystagmus None.
Vertigo Yes
Past-pointing none
Falling not tested
Romberg not tested
Turning head to right not tested
Turning head to left " "
Attempt to overthrow " "

---

~~TURNING~~

~~To RIGHT~~
~~Amp.~~
~~Duration Sec.~~

~~To RIGHT~~
~~Shoulder from above to to~~

~~Nystagmus~~
~~Vertigo~~
~~Past-pointing~~

~~To LEFT~~
~~Amp.~~
~~Duration Sec.~~

~~To LEFT~~
~~Shoulder from above to to~~

~~Nystagmus~~
~~Vertigo~~
~~Past-pointing~~

---

### CALORIC

Douche RIGHT
Amp.
After 1 min. 25 sec.
Conjugate rolling both eyes to Right.

Douche RIGHT
Shoulder from above 6° to Right ~~to~~

Nystagmus No quick component
Vertigo Apparently none, by statement of patient
Past-pointing Present
Falling Absent

Head Back Instead of → there is conjugate deviation to Right.
Amp.
2° to Right to

Douche LEFT ↶
Amp. Large
After min. 50 sec.

Douche LEFT
Shoulder from above 6° to Left to

Nystagmus Normal, slightly exaggerated amplitude
Vertigo None apparently
Past-pointing Present
Falling Present

Head Back ← normal nystagmus
Amp. Large
4° to Left to

paralysis. The turning tests were not undertaken. The caloric tests gave the following data:

*Right Ear.*—(1) Stimulation of the right vertical canals produces a *conjugate rotary rolling* of both eyes to the right. There is no quick recoil of the eyes to the left. The "quick component" of the nystagmus is absent, whereas the "slow component" is present—instead of a full nystagmus, therefore, there occurs only this rolling of the eyes to the right.

(2) Vertigo almost entirely absent. This is made evident not only by the statement of the patient, who says that she experiences no dizziness, but also because there is absolutely no tendency to fall toward the right. There is, however, a slight past-pointing of the right arm to the right, which shows that vertigo is not entirely absent.

(3) Stimulation of the right horizontal canal, instead of producing a full rhythmic nystagmus, shows exclusively a marked *conjugate deviation* to the right. Both eyes were drawn to the right and remained in that position; there was no quick recoil to the left.

*Left Ear.*—(1) Vertical canals. A real nystagmus is produced; not only are the eyes drawn to the left, but there is present the normal quick component to the right.

(2) Vertigo slightly impaired but by no means absent, as there is a past-pointing of the right arm to the left and a falling to the left.

(3) The left horizontal canal produces a normal nystagmus; the slow component to the left and the quick component to the right are both normally present.

Douching each ear produced one very striking phenomenon—not the least nausea was produced and yet sudden projectile vomiting occurred. Incidentally, this shows that there is probably a direct pathway from the ear to the nuclei of the vagus and phrenic nerves—the two nerves concerned in vomiting; this vomiting was due to direct stimuli and was not secondary to a cerebral sensation of nausea.

The essential features shown by the caloric test on this patient are:

(1) The noticeable impairment of *vertigo* from all the semicircular canals shows that there is a block somewhere along the vestibulo-cerebello-cerebral pathways.

(2) The eyes are drawn in the proper direction by the ear-stimulation. This would indicate normal vestibulo-ocular pathways throughout the medulla oblongata and pons, as well as normal labyrinths and VIII nerves.

(3) The most striking phenomenon is the absence of the quick component of nystagmus to the left. The contrast is definite; the quick recoil to the right is present and the quick recoil to the left is absent.

The following report was made: "The ear-tests suggest a lesion of the cerebrum, on the right side exclusively, at a point between the right cerebral crus and the right cerebral cortex. The following appear normal—both labyrinths, both VIII Nerves, the medulla oblongata, pons and cerebellum."

Autopsy, a few days after admission, showed a large subcortical abscess in the right parietal region (Fig. 121).

### COMMENT

The following conclusions are suggested by this case:

(1) It must be remembered that the patient was able to look to the left *voluntarily,* and yet the quick component of the nystagmus to the left was absent. This suggests the probability of two different cerebral centres for eye movement—the one presiding over volitional eye movement and the other controlling the more or less automatic eye movements such as are represented in the quick component of nystagmus.

(2) The centre controlling the quick component of nystagmus to the *left* is probably located in the *right* cerebral hemisphere.

A point worthy of mention in this case is that the patient was very ill at the time of the examination; the turning tests were practically impossible and at least unwise, and yet the douching tests alone furnished sufficient data for the suggestions noted above. This case also demonstrates that the ear tests are of value in *cerebral* cases as well as in the diagnosis of lesions in the posterior fossa.

CASE 22.—Mr. Walter W., age 35. Patient was admitted to the Hospital of the University of Pennsylvania on December 15, 1915, complaining of pain over the right eye and on the right side of the head, associated with vertigo.

The trouble began one year ago; while asleep in bed he had a convulsion, in which his legs and arms were flexed and he bit his tongue. Shortly afterward he developed a headache, which recurred every two or three days; these headaches were dull and heavy, lasting several days at a time. He also noticed that for a few minutes one side of his body and face felt numb. The attacks of headache became progressively worse and after a period of two months became associated with vomiting. At this time he began to have pain behind the eyes and the feeling of numbness on the left side of the body recurred. Six months after the onset the headache changed to the back of the neck. He is under the impression that he staggers at times and that this staggering usually occurs to the left. Once in a while he drops things from his left hand. His vision began to fail three weeks ago. At times, especially when he has severe headaches, he has attacks of vertigo in which he seems to be turning to the left. His appetite is good and there are no symptoms referable to the gastro-intestinal tract, lungs or the cardio-renal systems. He has noticed no change in the size of his face, feet or head. Speech is apparently normal. He has no difficulty in writing. He has had no convulsions except the ones mentioned at the onset of the trouble.

## CHART XXXV A

Name Mr Walter W. Age 35 Date 12/15/1915
Address
Referred by Dr Chas. H. Frazier

DIAGNOSIS:

SUMMARY:

Complains of Headache and vertigo

HISTORY:
Dizziness Yes
Staggering Yes
Deafness No
Tinnitus No

NOSE: Negative

THROAT: Negative

EARS:
A. D. Normal
A. S. Normal

Fistula Not tested

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 1 | A | 8 | Ac < Bc = n | Pol. | – | $c^4$ | Good | Galt | .8 | Weber |
| | 1 | | 8 | Ac < Bc = n | | – | | Good | | .8 | ± |

## CHART XXXV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None
Left external rectus – slight weakness

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling
Romberg Falls to Left
Turning head to right Not tested
Turning head to left " "
Attempt to overthrow Pushing to Left patient falls markedly to Left.

---

### TURNING

To RIGHT →
Amp. Large
Duration 33 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 30" to Right | 12" to Right<br>Left arm continues to past-point, long after right has ceased |

Nystagmus Exaggerated
Vertigo Exaggerated
Past-pointing Exaggerated

To LEFT ←
Amp. Large
Duration 31 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 36" to Left<br>Right arm continues To past-point long after the Left has ceased | 24" to Left |

Nystagmus Exaggerated
Vertigo Exaggerated
Past-pointing Exaggerated

---

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 45 sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 5" to Right | 8" to Right |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | RIGHT | LEFT |
|---|---|---|
| Head Back not tested<br>Amp. | to | to |

Douche LEFT ↶
Amp. Good
After min. 30 sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 6" to Left | 5" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | RIGHT | LEFT |
|---|---|---|
| Head Back Not tested<br>Amp. | to | to |

He denies venereal infection. Is married; his wife and two children are living and well.

The following are the notes of the physical examination made on his admission to the hospital: "The patient walks with a somewhat spastic gait and appears to be weak in the legs. There is only slight swaying when testing his station, not sufficient to call it a Romberg. There is marked spasticity in the left arm. The biceps and triceps jerks in the left arm are exaggerated. There is a suggestion of ataxia in the finger-to-nose test on the left side. Knee jerks are exaggerated on both sides, markedly so on the left and only slightly on the right. There is a distinct ankle clonus on the left side and a suggestion of a Babinski. On the right there is a slight non-persisting ankle clonus and the plantar reflexes are normal. Sensation is normal throughout the entire body. No asteriognosis in either hand. There is no paralysis of the muscles of mastication or of facial expression. Swallowing is normal. The tongue protrudes in midline. Taste is normal. The sense of smell is slightly impaired.

Examination of the heart, lungs and abdomen reveals nothing abnormal.

The Wassermann test of the spinal fluid is negative.

Eye examination showed slight papillœdema.

The ear examination was made on December 15, 1915, and the data are recorded on the accompanying chart (XXXV).

It is to be noted that there is no impairment or absence of any of the responses to ear stimulation either by turning or douching. This would suggest an intact vestibular apparatus. The turning tests, however, brought out markedly exaggerated nystagmus, vertigo and past-pointing, which would indicate a general irritation of the vestibular pathways concerned in the production of nystagmus and vertigo. The following report was made: "The ear examination suggests that the subtentorial region is affected by pressure only, from above or anteriorly, and that there is no lesion within the medulla oblongata pons or cerebellum themselves. The labyrinth and VIII Nerves are normal."

The patient was operated on by Dr. Charles H. Frazier on January 13, 1916, and an inoperable sarcoma of the right motor region was found.

Case 23.—Ida B., age 30. Was admitted to the Mt. Sinai Hospital on June 22, 1916, complaining of pain and tenderness about the left ear, left frontal headache, pain on the left side of the neck when turning the head to the right, and fever.

The patient was in perfect health until six weeks before admission, when she was taken with chills and fever just as she was getting over a "cold." At the same time she developed an "ear-ache," for which she applied to the dispensary for treatment. The drum-head was incised and the ear has been discharging ever since. Three days before admission to the hospital she developed a great deal of pain and tenderness over the left mastoid area with frontal headache and another rise of temperature.

The general physical examination at the hospital was practically negative, with the exception that she was dull and mentally apathetic. She looked pale and waxy and considerably beyond the age she stated, namely, 30. There was no weakness in either extremity. Sensation was normal throughout the body. All the reflexes were normal. The teeth were bad and the gums exuded pus. Chest and abdomen negative.

In eye examination by Dr. La Fever showed the media to be clear, the veins very tortuous, the cups closed but no gross changes. Myosis was unequal, the reactions being more than normal. There was a suggestion of a beginning optic neuritis.

The urine examination revealed a trace of albumen and many hyaline casts.

The blood shows 9400 leucocytes and 70 per cent. poly-morphonuclear cells.

The day following her admission to the hospital the pulse became slow and the temperature rose to 101°. The patient was restless during the night and she had auditory hallucinations.

The accompanying chart (XXXVI) contains the data of the vestibular examination.

It will be noted that stimulation of both horizontal semicircular canals produced practically normal nystagmus and vertigo with past-pointing and falling. Stimulation of the vertical semicircular canals of each ear produced no reaction whatsoever.

The hearing was normal on the right side and was impaired on the left. This deafness, however, was of the middle ear type, the left cochlea being normal.

There was a considerable amount of purulent discharge in the left ear which exuded with every pulse beat through an opening in the drum-head. The superior posterior wall of the external auditory meatus was boggy and depressed. The mastoid was edematous and tender.

It was evident that we were dealing in this case with more than an ordinary mastoiditis. The varying pulse, the rising temperature, the general apathy and listlessness, the frontal headache and hallucinations—all suggested that we were dealing with some form of an intracranial involvement complicating the mastoiditis. The microscopic findings of the spinal fluid as well as the entire clinical picture excluded meningitis. The low differential cell count and the absence of chills and fever made thrombosis of the lateral sinus extremely improbable. The most likely condition was a collection of pus within the cranial cavity. The diagnosis resolved itself into a differentiation between a cerebellar and a tempero-sphenoidal lobe abscess. The cerebellum was excluded as the seat of the abscess by the fact that stimulation of the labyrinth produced normal past-pointing of both arms in both directions. The exclusion of the cerebellum naturally pointed to the tempero-sphenoidal lobe as the seat of the abscess. This was further corroborated by the absence of reactions on stimulating the vertical canals of each ear. Normal hearing and normal reactions from the horizontal canals indicated that the labyrinths themselves were normal. With normal vertical semicircular canals the absence of responses to stimulation had to be accounted for by an interference with their nerve-pathways within the brain-stem. These fibres are found within the floor of the IV ventricle, and any increase of pressure in that region could easily interfere with the function of these pathways. An abscess in the tempero-sphenoidal lobe could produce such an increase of pressure within the IV ventricle.

Operation by Drs. Fisher and Watson revealed a necrotic mastoid with involvement of the tegmen antræ. Another incision was made in the scalp above the left ear parallel to the temporo-sphenoidal fissure and the temporo-sphenoidal

## CHART XXXVI A

Name Ida B. Age 30 Date 6/24/1916

Address

Referred by Mt. Sinai Hospital

DIAGNOSIS:

SUMMARY:

Complains of Pain behind and above Left ear. Left frontal headaches. Pain in Left side of neck.

HISTORY:

Dizziness Yes.

Staggering No.

Deafness Yes, left ear.

Tinnitus Yes, left ear.

NOSE: Negative

THROAT: Purulent gingivitis

EARS:

A. D. M. T. cloudy somewhat. Otherwise negative.

A. S. M. T. bulging. Superior canal wall slightly depressed. Marked tenderness all over mastoid region.

Fistula Not tested

Hearing Tests

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| A | 1 | A | 6 | Ac > Bc = n | Pol. | $c^4$ | Good | Galt | —— |
| = | 1 | | 4 | Ac < Bc > n | | | Good | | Weber |

## CHART XXXVI B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT ← slight
Looking to LEFT None
Looking UP ↑
Looking DOWN slight, more marked in left eye than Right

Shoulder from above

Nystagmus Yes
Vertigo No
Past-pointing No
Falling
Romberg No swaying.
Turning head to right Not tested
Turning head to left Not tested
Attempt to overthrow Not tested

POINTING

| RIGHT | LEFT |
|---|---|
| Touched | Touched |

---

### TURNING

To RIGHT →
Amp. Good
Duration 30 Sec.

To RIGHT
Shoulder from above

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

| RIGHT | LEFT |
|---|---|
| 24" to Right | 8" to Right |

very long duration

To LEFT ←
Amp. Good
Duration 30 Sec.

To LEFT
Shoulder from above

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

| RIGHT | LEFT |
|---|---|
| 12" to Left | 8" to Left |

Rather prolonged duration but not so long as to the Right

---

### CALORIC

Douche RIGHT None
Amp.
After 4 min. sec.
Putting head back there immediately appeared →

Douche RIGHT
Shoulder from above

Nystagmus Absent
Vertigo Absent
Past-pointing Practically absent
Falling

Head Back →
Amp. Good
Putting head forward

| RIGHT | LEFT |
|---|---|
| 2" to Right | Touched |
| 4" to Right | 6" to Right |
| 4" to Left | 3" to Left |
| 6" to Left | 4" to Left |

Douche LEFT None
Amp.
After 4 min. sec.

Douche LEFT
Shoulder from above

Nystagmus
Vertigo
Past-pointing
Falling

Head Back ←
Amp. Good
Head forward.

| RIGHT | LEFT |
|---|---|
| 8" to Left | Touched |
| 8" to Right | 4" to Right |

lobe exposed. The brain was probed in several directions but no pus found. On the following day, however, there was a large quantity of pus on the dressing, which evidently had made its way through the opening made at the time of operation. After a very stormy convalescence the patient made a complete recovery.

### COMMENT

This case well illustrates the value of the examination of the vestibular apparatus in cases of ear suppuration with intracranial symptoms. The clinical picture, while excluding many forms of intracranial complication, distinctly pointed to an abscess. The clinical symptoms themselves, however, gave no clue as to its definite location; it could have been in the cerebellum just as well as in the tempero-sphenoidal lobe. Since the ear suppuration was on the left side a strong point against tempero-sphenoidal lobe involvement was the absence of aphasia. The presence of hallucinations was of no great diagnostic value, since we had no means of knowing whether the woman was sane or insane (no history was obtainable from her or from any of her relatives which could have shed any light on this subject). The chances are that the physician would have operated over the tempero-sphenoidal lobe anyway simply for the reason that otitic abscesses are more frequent in that portion of the brain, but at best it would have been only a good guess.

CASE 24. -Emil K., age 14. Patient was admitted to the Jefferson Hospital on August 9, 1915, to the service of Drs. Dercum and George E. Price (through whose courtesy this case is reported), complaining of a paralysis of the left side.

Family history negative.

Patient was in excellent health until six years ago, when it was noted that he could not raise his left arm from the side. This partial loss of function of the left arm persisted until two years ago, when he was operated on at the Episcopal Hospital with the hope of restoring function. This was partially successful, but in a short time the condition relapsed and for the past year and a half it was gradually becoming worse. His left leg became affected four weeks ago, and he has been unable to walk since then. For the past two weeks he has vomited almost every day, especially in the morning. The following is a report of the physical examination made on admission to the hospital: "Well nourished and developed boy. Some exophthalmus of the left eye. Pupils unequal. On showing the teeth the mouth is drawn to the right side. The tongue is protruded toward the left. Heart, lungs and abdomen normal. Paralysis of the left upper and lower extremities. Both knee jerks increased. Double Babinski."

Blood examination normal. Wassermann negative.

*X-ray Report.*—Suggests internal hydrocephalus; the clinoid processes are ill-defined.

*Eye Examination.*—Reaction to light and convergence. O. D., none. O. S., slightly to light, but not to convergence. There are coarse nystagmic movements when the effort is made to converge. There is slight dissociation of ocular movements, although the binocular function is maintained. Rotation is limited in all directions, but more sharply limited upward and downward.

*Ophthalmoscopic.*—O. D. media clear; the disc margins are blurred; the cup is filled in; nerve markedly pale on the temporal side. Veins are tortuous but not full, the arteries are slightly contracted and there are no hemorrhages in the retinal field. O. S. media are clear; disc margins are hazy; disc pale throughout; the cup is filled in. The arteries are contracted. Pigment is irregularly distributed in the fundus, no white spots in retina. The atrophy of the nerve is more marked in the left than in the right eye.

*Diagnosis.*—Optic atrophy and a partial ophthalmoplegia.

The findings of the ear examination are recorded on the accompanying chart (XXXVII). It will be noted that there was no interference with or absence of any of the responses from stimulation of both ears by turning and douching. This would indicate an intact vestibular apparatus throughout. The only abnormality noticed were hyperactive responses in nystagmus, vertigo and past-pointing. The following report was made:

(1) Both labyrinths and VIII Nerves are normal. The impaired hearing in the left ear is not due to involvement of the labyrinth or the VIII Nerve, but to a chronic catarrhal change in the left middle ear.

(2) The medulla oblongata and pons (the brain stem) would appear to be normal because of the normal nystagmus obtained on stimulation of the horizontal and vertical semicircular canals of both ears.

(3) The right cerebellar hemisphere would appear normal because the right arm past-pointed correctly both to the right and the left after stimulation.

(4) The left cerebellar hemisphere could not be tested because of the paralysis of the left side of the body.

(5) The fact that the left VIII Nerve is normal would strongly suggest that there is no tumor in the left cerebello-pontile angle.

(6) All the responses in nystagmus, vertigo and past-pointing on stimulation of both ears were strikingly hyperactive which would suggest an irritation of the posterior fossa contents by some lesion above or anterior to the tentorium.

A decompression operation was done without revealing any tumor.

The patient died and the autopsy report by Dr. E. H. Funk was as follows: "A roughly oval tumor about 8 c.m. by 6 c.m. by 6 c.m. springs from the inner surface of the right hemisphere of the brain. It is rather firmly attached by its under surface to the corpus callosum and by its outer surface to the wall of the lateral ventricle. Its upper and inner surfaces are smooth and at points cystic in consistency. The inner surface of the skull is quite rough and irregular and appears to be thicker at some points than at others. The dura is normal in appear-

## CHART XXXVII A

Name Emil K. Age 14 Date 8/22/1915
Address
Referred by Dr Geo. E. Price

DIAGNOSIS:

SUMMARY:

Complains of Paralysis of left side

HISTORY:
Dizziness None
Staggering Yes
Deafness None
Tinnitus None

NOSE: Negative

THROAT: Negative

EARS:
A. D. Negative
A. S. O.M.C.C. Impaired mobility M.T.

Fistula

Hearing Tests

| $\underline{\underline{A}}$ | A | Ac | Bc | n | Pol. | $c^4$ | Galt |
|---|---|---|---|---|---|---|---|
|  |  | Ac | Bc | n |  |  |  |

A.D. Hearing normal.

A.S. Impaired hearing, entirely of the obstructive type.
Both cochleas appear normal.

## CHART XXXVII B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP None
Looking DOWN None
Rotation limited in all directions, especially upward and downward.

| POINTING | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Unable to test because of paralysis |

Nystagmus None
Vertigo None
Past-pointing None
Falling Not tested
Romberg Not tested
Turning head to right Not tested
Turning head to left Not tested
Attempt to overthrow Not tested

---

### TURNING

To RIGHT →
Amp. Large
Duration 33 Sec.

| To RIGHT | | |
|---|---|---|
| Shoulder from above | 24" to Right<br>18" " Right<br>Continued unusually long | to |

Nystagmus Exaggerated
Vertigo "
Past-pointing "

To LEFT ←
Amp. Large
Duration 32 Sec

| To LEFT | | |
|---|---|---|
| Shoulder from above | 20" to Left<br>16" Left<br>Continued unusually long | to |

Nystagmus Exaggerated
Vertigo "
Past-pointing "

---

### CALORIC

Douche RIGHT ↷
Amp. Good
After min. 45 sec.

| Douche RIGHT | | |
|---|---|---|
| Shoulder from above | 8" to Right | to |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | | |
|---|---|---|
| Head Back Not tested | to | to |

Amp.

Douche LEFT ↶
Amp. Good
After min. 42 sec.

| Douche LEFT | | |
|---|---|---|
| Shoulder from above | 8" to Left | to |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal
Falling Normal

| | | |
|---|---|---|
| Head Back Not tested | to | to |

Amp.

ance. The pituitary body was torn, but showed no gross lesion. The sella turcica is normal and the cerebral vessels are normal (Figs. 122 and 123).

Case 25.—Arthur P., age 25. The patient was referred to the hospital of the University of Pennsylvania, from St. Paul, Minn., with a diagnosis of a cerebellar lesion. He was admitted to the hospital on January 3, 1916, with the chief complaint of headache, vertigo and failing vision.

Illness began four months before admission with a sudden flashing of colors before the eyes, accompanied by headache, which lasted over night. These symptoms reappeared a week later. The headache began to come on with greater frequency and lately he has had a headache every day. The headache extends across the frontal region through the temporal bone to the occipital region, even down to the back of the neck. During the past month he has had double vision, also vertigo on arising in the morning, which would last for about an hour. Very frequently when the headache is very severe it is accompanied by vertigo. During the past month patient also finds it very difficult to walk in a straight line. He has been too weak to be about of late and he is mostly confined to bed. Several weeks ago there appeared vomiting spells which had no relation to ingestion of food. The stomach contents have occasionally shown traces of blood.

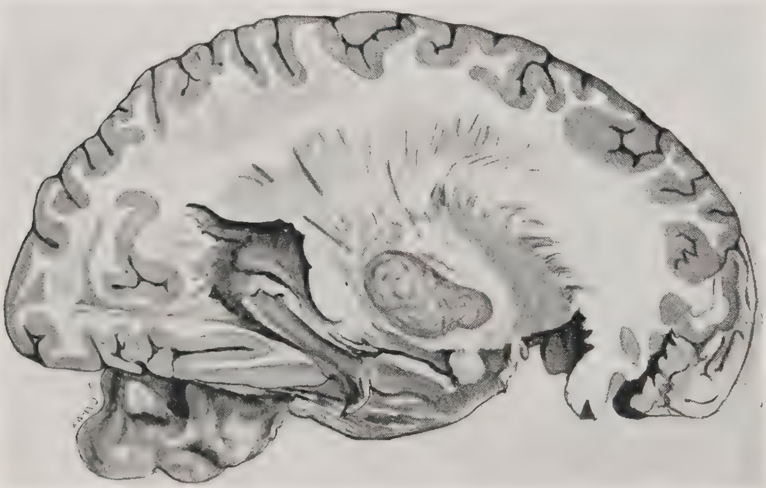


Fig. 122.—Tumor of corpus callosum.

The patient's brother stated that 15 years ago the patient received a severe blow in the centre of his forehead which rendered him unconscious for several hours. The brother also thinks that the patient is losing his memory and mental power.

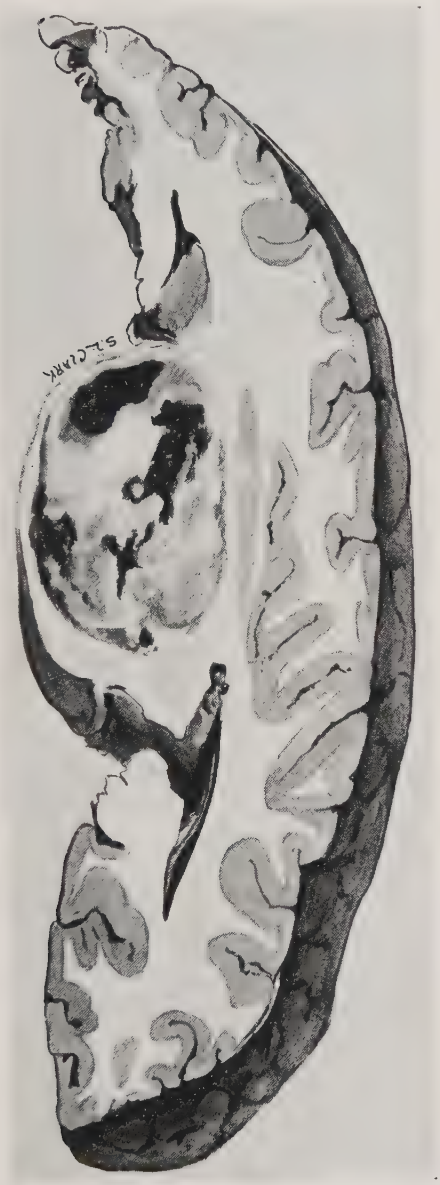


Fig. 123.—Tumor of corpus callosum.

## CHART XXXVIII A

Name Arthur P. Age 25 Date Jan. 5, 1916
Address
Referred by University Hospital

DIAGNOSIS:

SUMMARY:

Complains of Vertigo, flashes of light, headache.

HISTORY:
Dizziness Yes
Staggering Yes
Deafness None
Tinnitus None

NOSE: Not examined

THROAT: Not examined

EARS:
A. D. Much wax, but M.T. normal
A. S. Same except posterior third of M.T. is thin.

Fistula Not tested

Hearing Tests

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | 2 | A | 7 | Ac < Bc = n | Pol. | | $c^4$ | Good | Galt | .8 | Weber |
| | 2 | | 7 | Ac < Bc = n | | | | Good | | .8 | ± |

Hearing normal

## CHART XXXVIII B

### TESTS OF THE VESTIBULAR APPARATUS

SPONTANEOUS

NYSTAGMUS

Looking to RIGHT None
Looking to LEFT None
Looking UP ↑
Looking DOWN None

POINTING
RIGHT | LEFT

Shoulder from above 5" to Right | 1" to Right Touched

Nystagmus Vertical upward
Vertigo at times
Past-pointing Right arm to Right
Falling None
Romberg Some swaying.
Turning head to right does not increase swaying.
Turning head to left " " " "
Attempt to overthrow Pelvic girdle reaction normal.

---

TURNING

To RIGHT →
Amp. Large
Duration 34 Sec.

To RIGHT
Shoulder from above 36" to Right | 12" to Right.

Nystagmus somewhat exaggerated
Vertigo exaggerated
Past-pointing tremendous and very long duration.

O the left external rectus moves poorly -
Larger nystagmus in Right eye than Left.

To LEFT ←
Amp. Large
Duration 28 Sec.

To LEFT
Shoulder from above 36" to Left | 24" to Left

Larger nystagmus in Left eye than in Right.

Nystagmus somewhat exaggerated
Vertigo exaggerated.
Past-pointing tremendous and very long duration.

---

CALORIC

Douche RIGHT None
Amp.
After 4 min. sec.

Douche RIGHT
Shoulder from above 3" to Right | 4" to Right.

Nystagmus None
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back →
Amp. Large

8" to Right | 10" to Right.

Douche LEFT ←
Amp.
After 1 min 10 sec.

Douche LEFT
Shoulder from above 12" to Left | 12" to Left

Nystagmus perverted
Vertigo Normal
Past-pointing exaggerated
Falling Normal

Head Back ←
Amp. Large

12" to Left | 12" to Left

Previous medical history practically negative. Denies specific infection.

*Physical Examination.*—Patient fairly well nourished. No facial paralysis or disturbance of sensation. Reflexes everywhere present and apparently normal. Finger-to-nose test negative. Sense of smell undisturbed. No ankle clonus or Babinski. Extremities normal, examination of chest and abdomen negative.

Wassermann reaction negative. Lumbar puncture shows the spinal fluid under pressure of 15 m.m. of mercury. The fluid is clear.

*Eye Examination by Dr. de Schweinitz.* Pupils respond to ordinary stimuli. There is distinct limitation of the outward movement of each eye. No hemianopsia. Right eye: there is an exceedingly vascular choked disc of 4½ diopters. Many of the hemorrhages suggest the presence of thrombi. Left eye: the disc is choked but much less elaborately; the upper, lower and nasal edges are thinned, the thickness of the disc is about 3 diopters. There are no hemorrhages.

Examination of urine and blood are negative.

An examination of the vestibular apparatus was made on January 5, 1916, and the findings recorded on chart (XXXVIII). It will be noted that the hearing is normal in both ears.

(1) The presence of a spontaneous vertical nystagmus upward suggests an interference within the brain-stem.

(2) The spontaneous past-pointing of the right arm suggests a right cerebellar disturbance.

(3) The absence of nystagmus on douching the right ear with the head in the "so-called" upright position indicates an interference with the nerve-pathways for eye-movement within the right pons.

The most striking feature of the entire examination is the marked past-pointing of each arm to the right and left. This suggests that the cerebellum is intact. The exaggerated past-pointing, however, could only be accounted for by a supratentorial lesion irritating the cerebellum by pressure.

Pressure from such a lesion could easily account for the absence of nystagmus when the right vertical semicircular canals were stimulated, also for the perverted nystagmus from the left vertical canals as well as for the paretic condition of the external recti muscles.

The following report was made: "The ear examination suggests normal labyrinths and VIII Nerves, normal cerebellum and brain-stem. The hyperactive responses, chiefly those in past-pointing, suggest pressure upon the cerebellum from above the tentorium."

Patient had a decompression operation with a puncture of the corpus colossum on January 8, 1916. Following this the patient was improved and went home. The patient died on February 8, and his family physician reported that the autopsy showed a tumor situated on the left side of the posterior apex of the occipital lobe of the cerebrum. There was considerable destruction of the brain-tissue, and the cavity had worked forward and had almost broken into the left ventricle.

Case 26.—Mr. J. H. M., age 58. The patient was admitted to the University Hospital on June 19, 1915, with the chief complaint of staggering, vomiting and tinnitus.

The trouble dates back four years, when he began to have a buzzing in his left ear, following which the hearing became considerably impaired. This impairment of hearing progressed steadily and quite rapidly, so that now he is practically deaf. Last summer he began to stagger. He has never had any headache, but in the past six months he has vomited off and on. The vomiting is not preceded by nausea, but is followed by retching. He has been rapidly failing in the last two months, particularly in the last two weeks.

The previous medical history is negative.

He denies any venereal history.

*Physical examination.*—Station poor, falls toward the left. In walking he staggers both ways, but more to the left. Adiadokokinesis more marked with the left hand. Marked ataxia in finger-to-nose test with left hand especially. Some tremor and hypermetry with both hands. Sense of smell good. The V, IV, IX, X and XI cranial nerves appear to be uninvolved. There is a slight weakness possibly of the lower division of the right VII Nerve. There may be a slight weakness of the grip in the right hand. Sense of muscular position in upper extremities appears to be normal. Biceps jerks very good. Sensation present over entire body, although it seems to be slightly delayed. There is no spasticity of the lower limbs. Knee jerks prompt; no ankle clonus and no Babinski on either side.

Examination of chest, lungs, heart and abdomen negative.

*Eye examination by Dr. de Schweinitz* shows a choked disc of 6 diopters on the right side and 7 diopters on the left side. Pupils are equal, react normally to light and accommodation. There is no hemianopsia.

Examination of the urine and blood negative.

Examination of the vestibular apparatus was made on June 21, 1915, and the findings recorded on accompanying chart (XXXIX).

It will be noted that the hearing in the right ear is normal; the left ear is stone deaf. Among the spontaneous phenomena we have:

(1) Marked nystagmus on looking to the right.
(2) Weakness of the right and left internal recti.
(3) A spontaneous past-pointing of the right arm to the left.
(4) Spontaneous falling to the left with a stiff pelvic girdle.

The turning test suggests a non-functionating left labyrinth because the nystagmus after turning to the right lasted 16 seconds, whereas after turning to the left it was of 23 seconds' duration. There was also an absence of past-pointing of the left arm to the left.

Douching the right ear produced prompt responses from the vertical semicircular canals as well as from the horizontal canal. The nystagmus, however, was *inverse;* that is, it was in a direction opposite to what it ought to be, namely, to the right instead of to the left. The nystagmus was also perverted, that is the wrong kind—horizontal instead of rotary—when the vertical canals were stimulated.

Douching the left ear produced practically no responses at all.

In attempting to analyze these findings due attention must be paid to the differentiation between a central and peripheral lesion. The chief complaint in this case, namely, staggering, which was ushered in by tinnitus and gradually increasing deafness, would suggest a peripheral lesion. This would appear to be corrobo-

## CHART XXXIX A

Name Mr. J. H. M. Age 58 Date June 21, 1915.
Address
Referred by University Hospital

DIAGNOSIS:

SUMMARY:

Complains of Staggering, tinnitus, deafness, vomiting

HISTORY:
Dizziness Yes
Staggering Yes
Deafness Yes
Tinnitus Yes

NOSE: Not examined

THROAT: Not examined

EARS:
A. D. Impaired mobility of hammer handle.
A. S. Impaired mobility of hammer handle,

Fistula Not tested

Hearing Tests

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| $\underline{A}$ | 1 | A | 5 | Ac | < | Bc | > | n | Pol. | | $c^4$ | Fair | Galt | Weber |
| = | 0 | | 0 | Ac | 0 | Bc | 0 | n | | | | 0 | | $\underline{+}$ |

With noise apparatus in Right ear cannot hear loud shouting.

## CHART XXXIX B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT ↶ marked
Looking to LEFT None
Looking UP ↶
Looking DOWN None

Apparent weakness of both ext. recti

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 2" to Left | Touched |

Nystagmus Rotary on looking upward and to Right.
Vertigo None
Past-pointing Right arm to Left
Falling to Left
Romberg falls to Left even with eyes open; more so with eyes closed.
Turning head to right tendency to fall forward and to Left.
Turning head to left " " " " " " "
Attempt to overthrow poor pelvic girdle movements in all directions.

---

### TURNING

To RIGHT →
Amp. Good
Duration 16 Sec.

To RIGHT

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 6" to Right | 12" to Right (long duration) |

Nystagmus practically normal, although shortened
Vertigo Impaired
Past-pointing Normal

To LEFT ←
Amp. Good - somewhat irregular
Duration 23 Sec.

To LEFT

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 12" to Left | Touched |

Nystagmus Normal
Vertigo Normal
Past-pointing absent of Left arm

---

### CALORIC

Douche RIGHT ←
Amp. Good
After min. 35 sec.

Douche RIGHT

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | 6" to Right | 6" to Right |

Nystagmus perverted and inverse
Wrong direction and kind
Vertigo Normal
Past-pointing Normal
Falling Normal

Head Back ← wrong direction
Amp. Large

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Head Back | 7" to Right | 24" to Right |

Douche LEFT
Amp.
After min. 46 sec.

Douche LEFT

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Shoulder from above | Touched | 2" to Right |

Nystagmus practically absent
Some irregular movements
Vertigo absent
Past-pointing Absent
Falling Absent

Head Back Irregular twitchings is all that is produced.
Amp.

| | POINTING RIGHT | POINTING LEFT |
|---|---|---|
| Head Back | Touched | Touched |

rated by the tests, which show a distinct involvement of one ear. This case, however, is one of central lesion, because

(1) The unsteadiness of gait and spontaneous nystagmus were scarcely noticeable at the beginning of his illness, but gradually became worse as time went on. This is not the rule in a labyrinthine disturbance, in which the onset of the

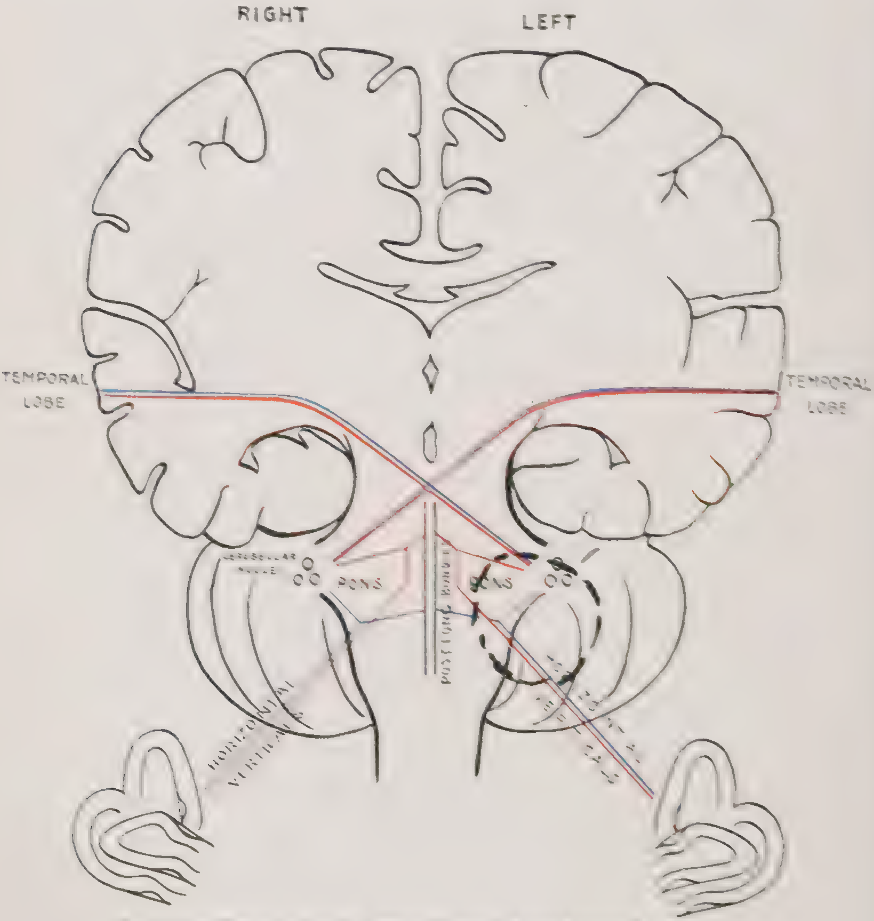


Fig. [illegible] pons.

various phenomena, such as staggering and nystagmus, is very marked and explosive, reaching a climax very quickly and then gradually subsiding.

(2) The nystagmus obtained after douching was "oblique," and "perverted." This always indicates a disturbance of the brain-stem.

(3) The past-pointing after turning and douching was very much more

marked in one arm than in the other. Obviously this could not be produced by an "end organ" disturbance.

It must also be noted, however, that douching the left ear produced practically no response. This ear was also stone deaf, which would indicate a destruction of the labyrinth or the VIII Nerve of this side. This case, therefore, presents the picture of a central lesion with the destruction of the left "end-organ." Such a combination is best explained by a lesion in the cerebello-pontile angle (Fig. 124).

The question of actual involvement of the neighboring structures, namely the brain-stem or cerebellum, is, of course, important. We note on the chart that the left arm fails to past point to the left under all circumstances. This would suggest involvement of the left cerebellar hemisphere. There were other examinations made in this case on different dates, however, and at times the left arm did past-point to the left. This would make an actual involvement of the cerebellar substance improbable; the failure of response as noted is therefore probably due to pressure. The medulla oblongata and pons are most likely actually involved by the lesion because:

(1) The vertical semicircular canals of the right or opposite side react, which indicates that the angle lesion is not exerting any great amount of pressure against the brain-stem. Any evidence of brain-stem disturbance, therefore, is probably due to a lesion of the brain-stem itself and not produced by pressure from without.

Douching the right ear produced both an "inverse" and "perverted" nystagmus.

The patient was operated on and the following report made by the surgeon: "The tumor was about as large as a hen's egg, situated in the cerebello-pontile angle on the right side and was adherent by a broad base in the region of the pons."

The above report was sent to us in a letter written by Dr. George P. Müller, who operated on the case. When this letter was received stating that a tumor was found on the right side we were rather chagrined to think that the ear tests could be so misleading. We then wrote to Dr. Müller and jestingly suggested that he had operated on the wrong side; according to the ear examination the lesion was in the left cerebello-pontile angle and not the right. We were gratified to receive a prompt reply telling us that he had consulted the hospital records and found that he had made a mistake in his letter—that the lesion and operation were both on the left side as the ear report suggested.

Case 27. Mr. G. C. M., age 49. Patient was admitted to the Samaritan Hospital on April 19, 1915, with the history that he was unable to walk since the preceding August.

The trouble commenced about a year ago, when he became extremely nervous. At this time he complained of feeling generally weak, but had no pain and no headache. At times he had tremor and involuntary movement. Now, his speech is slow and labored. He is unable to write. Is extremely deaf, the left ear having failed seven years ago and the right ear two months ago.

*Physical Examination.*—General condition very good. Patient is unable to walk because of inco-ordination and not paralysis. Speech is slurring, indistinct and of the bulbar type. Facial movements are active on the left side, particularly about the mouth. Tongue protrudes to right side of middle line, but is freely movable. The left eye is not closed as quickly as the right. There is evidence of

## CHART XL A

Name Mr G. C. M. Age 49 Date May ,10, 1915.
Address
Referred by Dr. Samuel Ingham.

DIAGNOSIS:

SUMMARY:

Complains of Vertigo, inability to walk.

HISTORY:
Dizziness Yes
Staggering Yes
Deafness Yes, left ear
Tinnitus None.

NOSE: Septal deviation to Right.

THROAT: Negative

EARS:
A. D. Slightly retracted. Perforation anterior superior quadrant.
A. S. Slightly retracted. M. T. thickened.

Fistula Negative.

Hearing Tests

|  |  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|
| $\underline{\underline{A}}$ | A | Ac | Bc | n | Pol. | $c^4$ | Galt |
|  |  | Ac | Bc | n |  |  |  |

Unsatisfactory because of mental condition.
Statements contradictory While douching right ear shouted words could not be heard. While douching the left ear the patient answered questions.

## CHART XL B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT ← marked and rapid
Looking to LEFT slight → indefinite
Looking UP ↑ and to the Right
Looking DOWN ↓ and to the Right
Looking straight ahead slight ←

POINTING

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | 3" to Left<br>Left arm incoordinate, so that pointing is difficult. |

Nystagmus marked in all directions except to Left
Vertigo None
Past-pointing Left arm to Left
Falling to Right.
Romberg falling to Right.
Turning head to right
Turning head to left
Attempt to overthrow

### TURNING

Examination made when turning chair was not available.

To RIGHT
Amp.
Duration Sec.

| | RIGHT | LEFT |
|---|---|---|
| To RIGHT<br>Shoulder from above | to | to |

Nystagmus
Vertigo
Past-pointing

To LEFT
Amp.
Duration Sec.

| | RIGHT | LEFT |
|---|---|---|
| To LEFT<br>Shoulder from above | to | to |

Nystagmus
Vertigo
Past-pointing

### CALORIC

Douche RIGHT Changed the spontaneous to the Right into ↱↓×↔
Amp. Fair
After 1 min. 50 sec.

| | RIGHT | LEFT |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | Touched | 3" to Left<br>Same as spontaneous |

Nystagmus definite response although delayed.
Vertigo None
Past-pointing None
Falling None

Head Back →
Amp. Good

Vertigo marked

| | RIGHT | LEFT |
|---|---|---|
| Head Back | 2" to Right | 3" to Left<br>Same as spontaneous |

Douche LEFT No change
Amp.
After 4 min sec.

| | RIGHT | LEFT |
|---|---|---|
| Douche LEFT<br>Shoulder from above | Touched | 3" to Left<br>Same as spontaneous |

Nystagmus Absent
Vertigo Absent
Past-pointing Absent
Falling Absent

Head Back none
Amp.

| | RIGHT | LEFT |
|---|---|---|
| Head Back | Touched | 3" to Left<br>Same as spontaneous |

some loss of sensation and a feeling of numbness in the distribution of the left V Nerve. Muscular power of entire body seems good.

Corneal reflexes on both sides present. Tendon reflexes are generally active.

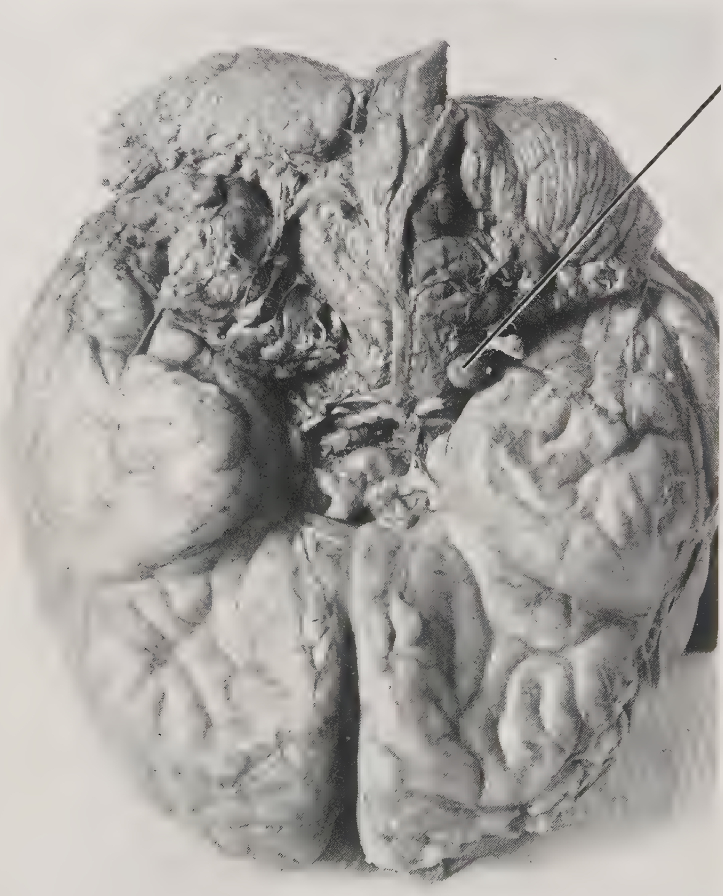


FIG. 125.—Brain showing tumor of the left cerebellar lobe and involving the cerebello pontile angle. About 1/3 of the inferior portion of the left cerebellar lobe has apparently been destroyed and the 5th, 7th and 8th cranial nerves are displaced and entangled in the mass of the tumor. Another small tumor is shown on the surface of the right lateral aspect of the pons near the cerebral peduncle.

Knee jerk and Achilles jerk are much increased. There is a unilateral ankle clonus and a right patellar clonus. Babinski present on the left side, but doubtfully on the right. Abdominal reflex diminished on both sides.

Co-ordination of right arm is fair. Finger-to-nose test and finger-to-finger test are defective. Movements of left arm are very inaccurate, the hand oscillating several inches about an object which he attempts to touch. There is marked incoordination of the left leg and not so much of the right. Has difficulty in swallowing liquids, the same at time regurgitating through the nose.

*Eye Examination.*—Right pupils slightly larger than left; both react to light and accommodation. There is a spontaneous nystagmus in every direction. No paralysis of extraocular muscles. There is a double choked disc of 6 diopters.

Wassermann negative. An examination of the vestibular apparatus was made on May 10, 1915, and the accompanying chart (XL) shows the data obtained. Although it was difficult to determine the hearing in this case because of the mental condition of the patient, nevertheless it was evident that he was stone deaf in his left ear, and that some hearing was present in the right ear.

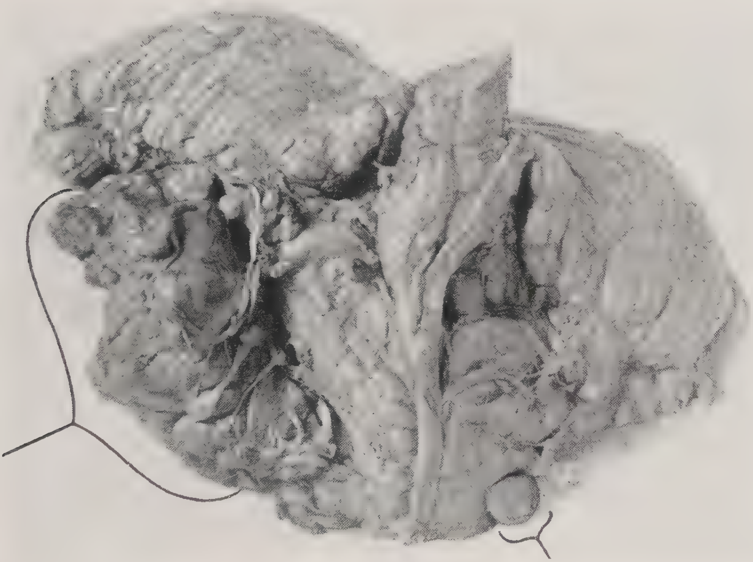


FIG. 126.—Same specimen as figure 125, showing only cerebellum and brain-stem which have been separated from cerebrum.

Douching the left ear failed to produce any response whatsoever. This, together with the total deafness on this side, indicates a destruction of the labyrinth or the VIII Nerve. Stimulation of the right vertical semicircular canals produced perverted nystagmus, which suggests an involvement of the pons by pressure or infiltration. Stimulation of the right horizontal canal produced a normal nystagmus and marked vertigo. In spite of the vertigo, however, the left arm failed to past-point to the right.

The salient features of this examination were:

(1) A central lesion.

(2) Destroyed left labyrinth or VIII Nerve.

(3) Absence of vertigo and past-pointing to all tests except douching the right ear with the head back—in other words, the only past-pointing that was elicited was of the right arm to the right.

A central lesion with a loss of all reactions from one ear indicated that the lesion must necessarily involve the cerebello-pontile angle of that side. The absence of vertigo and past-pointing in this case, however, was so marked that a cerebellar lesion had to be considered. The following report was made: "The ear examination would suggest a large lesion of the cerebellum, more marked on the left side, pressing into the left cerebello-pontile angle, and crowding the pons to the right, affecting the posterior longitudinal bundles either by infiltration or pressure."

Autopsy on May 25, 1915, by Dr. Wohl reads as follows: "A mass of considerable size was found anterior to the cerebellum on the left side, surrounding the cranial nerves in that vicinity and particularly adherent to the VIII Nerve at its point of emergence from the temporal bone. This mass was of a soft consistency and contained four small cysts." (Figs. 125 and 126.)

Case 28.—Mrs. Elizabeth R., age 40. Patient was referred by Dr. F. X. Dercum on April 25, 1915, for an examination of the vestibular apparatus. She gave the following history: One year ago she began to be troubled with dizziness. Very frequently these attacks of dizziness would assume the character of a definitely systematized vertigo, that is, objects about her or she herself would actually appear to be moving in one or the other direction. Eight months ago she began to stagger; this gradually became worse so that now she cannot walk straight at all. Five months ago vision began to fail. Three months ago patient suddenly became deaf in the right ear. The findings of the ear examination are given in the accompanying chart (XLI).

Notes of her physical examination were not available, but a study of the ear chart alone suggests that we are dealing with a central lesion because:

(1) Weakness of the right external rectus muscle.

(2) Turning to the left produced a fair past-pointing of the right arm to the left, whereas the left arm did not past-point at all. When a peripheral lesion interferes with normal past-pointing both arms are usually similarly affected.

(3) Turning the patient to the right produced a past-pointing of the right arm to the left—in the wrong direction. A peripheral lesion might prevent past-pointing, but is not likely to produce the wrong kind of past-pointing.

(4) Douching the left ear produced a very poor nystagmus from the vertical semicircular canals and a large nystagmus from the horizontal canal. This poor reaction from the left vertical canals could hardly be accounted for by a lesion of the canals themselves, because it is difficult to conceive of a labyrinthine disturbance which would markedly affect only one set of canals and leave the other canal entirely normal. We further note that stimulation of the right ear gave abso-

lately no responses. This, combined with total deafness on that side, indicates a destruction of the right labyrinth or right VIII Nerve—in other words, a peripheral lesion of the right side. This combination of a central and peripheral disturbance is easily explained by a lesion located in the cerebello-pontile angle.

Two days later another ear examination was made and the findings recorded on the accompanying chart (XLII). It will be noted that at this second examination the following differences were observed:

(1) There was now present a spontaneous vertical nystagmus upward.

(2) At this time there was a distinct weakness of *all* the ocular muscles.

(3) Douching the left ear with the head in the "so-called" upright position, which stimulates the left vertical semicircular canals, failed to produce any reaction. Evidently something had occurred in the past two days which interfered with the normal responses from the left vertical semicircular canals pathways.

The above changes simply corroborated our opinion that we were dealing with a lesion in the right cerebello-pontile angle. An increase of pressure by this lesion could well account for the sudden appearance of the spontaneous vertical nystagmus upward, the increasing weakness of the ocular muscles as well as for the abolition of function from the pathways of the left vertical semicircular canals.

The ear report was as follows: "There is a lesion in the right cerebello-pontile angle (Fig. 127). This lesion, however, originates probably in the brain-stem and involves the angle secondarily for the following reasons:

(1) The symptoms in this case date as far back as one year ago, whereas the deafness appeared only three months ago. In an acoustic tumor deafness would most likely be among the first symptoms.

(2) There is evidence in this case of the actual involvement of the posterior longitudinal bundles themselves, because the III and VI cranial nerves are considerably involved, and there is present a spontaneous nystagmus in every direction.

(3) There is an inco-ordination of associated ocular movements. When the left horizontal canal was stimulated the resulting nystagmus was different in each eye; the left eye showed a larger excursion than the right."

The patient was operated on by Dr. J. C. DaCosta and the right cerebello-pontile angle exposed. A large tumor was found which it was impossible to remove.

Two weeks later when the patient had sufficiently recovered from the operation, another examination of the vestibular apparatus was made and some very interesting changes noted.

(1) Signs of improved function: (*a*) Spontaneous ocular movements are less restricted; (*b*) Stimulation of the left vertical semicircular canals fibres now again produces responses; (*c*) On looking upward there is a less noticeable vertical nystagmus upward, and it has assumed a more rotary character.

(2) Signs of further impairment: (*a*) There is now a marked inco-ordination of the right arm; (*b*) The left arm does not point to the left when the left ear is douched in spite of a good nystagmus. This would suggest an extension of the lesion either into the cerebellum itself or an involvement of the nerve fibres on their way to or from the cerebellum.

## CHART XLI A

Name Mrs Elizabeth R. Age 40 Date April 25, 1915.
Address
Referred by Dr F. X. Dercum.

DIAGNOSIS:

SUMMARY:

Complains of Deafness, vertigo, staggering.

HISTORY:
Dizziness Frequent attacks during past year.
Staggering Began 8 months ago, - now marked.
Deafness Complete in right ear. Came on 3 months ago.
Tinnitus None

NOSE: Not examined

THROAT: Not examined

EARS:
A. D. M. T. retracted - no scar or perforation
A. S. M. T. retracted - no scar or perforation.

Fistula Not tested

Hearing Tests

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 0 | A | 0 | Ac | ? | Bc | ? | n | Pol. | – | $c^4$ | 0 | Galt | – Weber |
| = | 2 | | 8 | Ac | > | Bc | < | n | | | | Good | | to Left. |

Stone deaf right ear.
Normal hearing left ear

## CHART XLI B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT ← slight
Looking to LEFT → more marked
Looking UP None
Looking DOWN None

Suggestion of weakness of Right external static.

| POINTING | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus to Left on looking to Left
Vertigo None
Past-pointing None
Falling None
Romberg Negative
Turning head to right slight swaying.
Turning head to left " " .
Attempt to overthrow Pelvic girdle reactions normal

---

### TURNING

To RIGHT →
Amp. not fully normal but fair
Duration 10 Sec.

| To RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched<br>3" to Left | Touched on rapid turning.<br>½" to Right |

Nystagmus Impaired
Vertigo None
Past-pointing None

To LEFT ←
Amp. Large
Duration 9 Sec.

| To LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 4" to Left | Touched |

Nystagmus Impaired
Vertigo Normal
Past-pointing Impaired right, absent left arm.

---

### CALORIC

Douche RIGHT None
Amp. None
After 4 min. sec.

| Douche RIGHT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |
| Head Back | Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back None
Amp.

Douche LEFT ↶
Amp. Poor
After 1 min. 8 sec.

| Douche LEFT | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | 3" to Left | 3" to Left. |
| Head Back | 5" to Left | 3" to Left. |

Nystagmus Impaired
Vertigo Normal
Past-pointing Normal
Falling Normal.

Head Back ←
Amp. Large.

## CHART XLII

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

POINTING
RIGHT LEFT

Looking to RIGHT ← Shoulder from above very incoordinate but Touched ½" to Left.

Looking to LEFT → marked more than looking to Right

Looking UP ↖) and ↑

Looking DOWN None

Weakness of all ocular muscles

Nystagmus in every direction except on looking down.
Vertigo None
Past-pointing Practically none
Falling None
Romberg Slight swaying.
Turning head to right does not increase swaying.
Turning head to left " " " " .
Attempt to overthrow Pelvic girdle reaction normal

---

### TURNING

To RIGHT →
Amp. Fair
Duration 10 Sec.

To RIGHT
Shoulder from above Touched 3" to Left Touched ½" to Right

Nystagmus Shortened
Vertigo None
Past-pointing None

To LEFT ←
Amp. Large
Duration 10 Sec.

To LEFT
Shoulder from above 4" to Left Touched

Nystagmus Shortened
Vertigo Normal
Past-pointing of Left arm absent to Left

---

### CALORIC

Douche RIGHT None
Amp.
After 3 min. sec.

Douche RIGHT
Shoulder from above Touched Touched

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back
Amp.
None Touched Touched

Douche LEFT None
Amp.
After 3 min. 49 sec.

Douche LEFT
Shoulder from above Touched Touched

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back ←
Amp. Large Left eye moves more than the right. 5" to Left 5" to Left

## Comment

Clinical experience with cerebello-pontile angle lesions has frequently shown that the vertical semicircular canals of the side

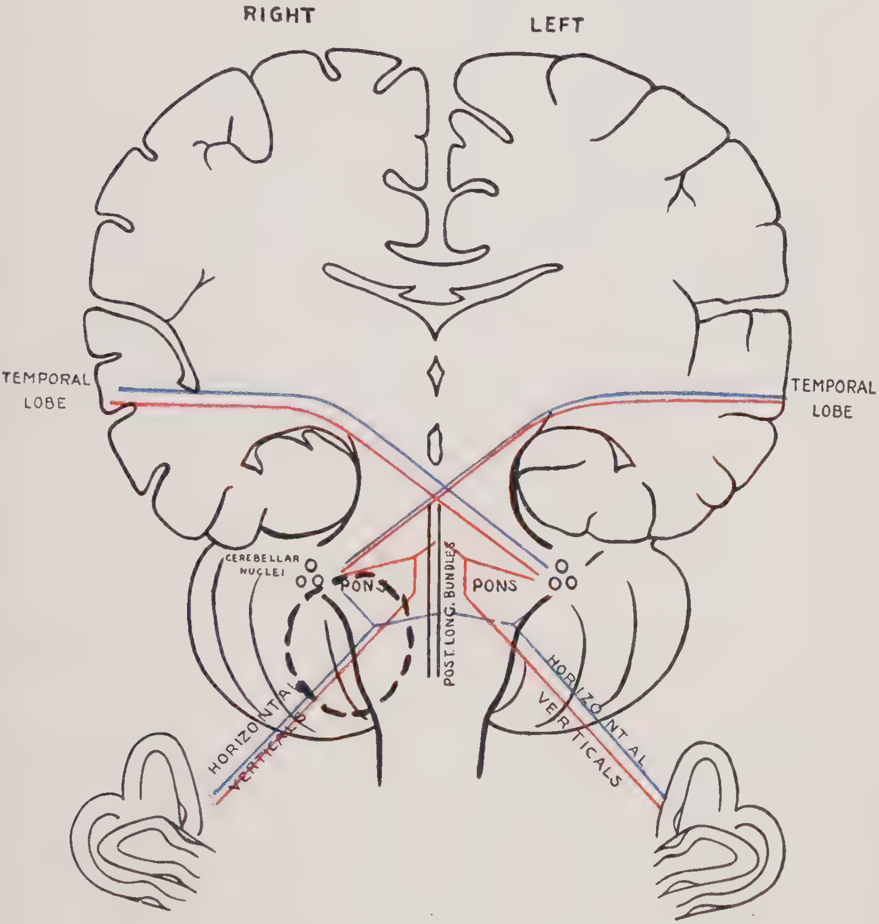


Fig. 127.—Large tumor in right cerebello-pontile angle.

opposite the lesion fail to respond to stimulation. This has occurred with such a uniformity that no phenomenon-complex appears to be complete without it. We had always supposed that it was due to pressure transmitted across the midline. This case demonstrates the truth of this assumption. The left vertical semicircular canals and pathways responded to stimulation on the first examination; two days later they failed to respond. After the decompression operation was done we were again able to obtain responses, which proves that there was no organic involvement of the fibres, but that they were simply affected by the increased pressure.

Case 29.—Miss Anna S., age 33. Patient was admitted to the University Hospital on June 2, 1916, with the chief complaint of "staggering" and "blind spells."

The trouble commenced three years prior to her admission to the hospital. The first thing she noticed was that she would become dizzy while dancing. Shortly after that she would become dizzy every time she turned her head to the right. A little later it was noticed that she could not walk in a straight line. The vertiginous attacks were associated with tinnitus. Examination at that time disclosed a right-sided deafness. She went along in fair comfort until about three months ago, when the "blind spells" became much more pronounced; they would come on more frequently and would last a longer period of time. The hearing becomes very much worse during those blind spells. At no time did she have headache or vomiting.

The previous medical history as well as the family history is negative.

The following is a report of the physical examination: "Patient is an adult female of small stature. The right side of the face seems to be slightly weaker. The patient is unable to lift the right corner of the mouth as well as the left. Patient is able to distinguish between "sharp" and "dull" over the right side, but the point of the pin does not feel as sharp on this side as it does on the left. When walking with the eyes closed she staggers slightly to the left—this is not marked. There is no ataxia; there is no weakness of the limbs; no Romberg; no ankle clonus; no Babinski; the knee jerks are slightly weak. Examination of chest and abdomen negative."

*Eye Examination by Dr. Shumway.*—Low grades of optic neuritis in both eyes. Height of the nerve is not over 3 diopters. Vision in the right eye, 6/12ths; in the left, 6/30ths. There is a concentric contraction of both fields, especially the left. Pupils react to light and accommodation. Extraocular muscles seem normal. There is a constant rotary nystagmus to the left, much increased by looking to the right or left.

X-ray examination negative.

The patient was under the care of a neurologist, who sent her to the University Hospital with the following report: "The history and the present condition all point to a cerebellar involvement. The involvement of the V and VIII Nerves of the right side speak in favor of a probable neoplasm in the right cerebello-pontile angle. In view of the gradual loss of vision we advised a subtemporal decompression operation."

An examination of the vestibular apparatus was made on June 2, 1916, and the findings recorded in the accompanying chart (XLIII).

The hearing tests demonstrated normal hearing on the left side and total nerve-deafness on the right side.

Without reference to the neurologic aspect of the case, the result of the ear examination would seem to suggest at first glance a labyrinthine lesion. The gradual onset of the deafness, for it must be recalled that it was noted as long as three years prior to this examination, the tinnitus which was always more marked during the "blind spells," the presence of a spontaneous rotary nystagmus which usually accompanies labyrinthine disturbances—all these would suggest a peripheral lesion. Furthermore, the shortened duration of the nystagmus after turning to the left would appear to bear out the theory of a destroyed right labyrinth. A careful study of the chart alone, however, would show that we are dealing with a central lesion because:

(1) The spontaneous eye movements were not the same in both eyes. The nystagmic movements in the right eye were more marked than in the left. This obviously could never be produced by a labyrinthine lesion.

(2) Turning to the right produced a good horizontal nystagmus, in the proper direction, of 24 seconds. This of itself would indicate the ability of the "end-organ" to generate normal impulses. We should therefore expect normal past-pointing. Instead of this the left arm failed to past-point to the right. Similarly, turning to the left failed to produce normal past-pointing of the right arm to the left—in other words, the past-pointing with both arms is outward, no matter what the direction of the turning. Such "crossed" past-pointing is explicable only on the basis of a central lesion.

(3) Douching the left ear with cold water produced a perverted nystagmus, which indicates a brain-stem disturbance.

A salient feature of this examination, however, is the total absence of all responses from the right side, which indicates a peripheral destruction of the right ear or all of its pathways. For a central lesion to involve the peripheral ear pathways it must be situated in the cerebello-pontile angle, where the VIII Nerve enters the brain-stem.

The following report was made: "A lesion of the right cerebello-pontile angle involving the cerebellar substance and probably inoperable."

The patient was operated on on June 17, 1916, and a large mass found in the right cerebello-pontile angle which it was impossible to remove.

The autopsy showed that anterior to the right cerebellar region there was an egg-shaped mass measuring 5 by 4½ by 3 c.m. It was of the same color as the rest of the brain, of rather firm consistency, definitely encapsulated and apparently not attached to the cerebellum. The right cerebellar lobe itself had lost most of its characteristics. It was rather soft, somewhat cystic with indistinct striations. The microscope showed the specimen to be a glioma. The hypophysis was normal.

## CHART XLIII A

Name Miss Anna S. Age 33 Date June 2, 1916.
Address
Referred by University Hospital

DIAGNOSIS:

SUMMARY:

Complains of Dizziness and blind spells.

HISTORY:
Dizziness Yes
Staggering Yes
Deafness Yes
Tinnitus Yes

NOSE: Negative

THROAT: Negative.

EARS:
A. D. O.M.C.P.H. Entire posterior half cicatricial. Hammer handle immovable
A. S. Slightly retracted, slight scar back of umbo.

Fistula Not tested

Hearing Tests

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | ○ | A | ○ | Ac ○ | Bc ○ | n | Pol. | .6 | $c^4$ | ○ | Galt | .6 |
| = | 2½ | | 9 | Ac > | Bc < | n | | | | Good | | |

Stone deaf in Right ear.
Hearing normal in Left ear

## CHART XLIII B

### TESTS OF THE VESTIBULAR APPARATUS

#### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT ↷
more marked in Right eye. - large amplitude
Looking to LEFT ↷
Looking UP ↷
Looking DOWN None

Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| Touched | Touched<br>1" to Right |

Nystagmus Rotary to Left
Vertigo None
Past-pointing None
Falling None
Romberg No swaying.
Turning head to right Does not produce swaying
Turning head to left " " "
Attempt to overthrow Pelvic girdle reacts normally.

---

#### TURNING

To RIGHT →
Amp. Good
Duration 24 Sec.
Soon mixed with spontaneous ↷

To RIGHT
Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 16" to Right | 7" to Left<br>immediately after turning<br>8" to Left |

Nystagmus Good
Vertigo Slight
Past-pointing Left arm goes in wrong direction

To LEFT ←
Amp. Large
Duration 13 Sec.

To LEFT
Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 5" to Left<br>2" to Right | 18" to Left |

Nystagmus Large, but shortened.
Vertigo sub-normal
Past-pointing Right arm continues to past-point in the wrong direction while Left continues for 15 or 16 consecutive pointings

---

#### CALORIC

Douche RIGHT None
Amp.
After 3 min. sec.

Douche RIGHT
Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 2" to Right<br>Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back None
Amp.

| POINTING RIGHT | POINTING LEFT |
|---|---|
| Touched | Touched |

Douche LEFT ↶ and ←
Amp. Large
After min. 30 sec.

Douche LEFT
Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 2" to Right | 8" to Left |

Nystagmus Good but perverted.
Vertigo None
Past-pointing Right arm wrong direction.
Falling None

Head Back Immediately appeared large ← with
Amp. tendency to conjugate deviation to Left

| POINTING RIGHT | POINTING LEFT |
|---|---|
| Touched | 1" to Right |

## COMMENT

The neurologic data in this case was sufficient on which to base a diagnosis of tumor in the right cerebello-pontile angle (Fig. 128).

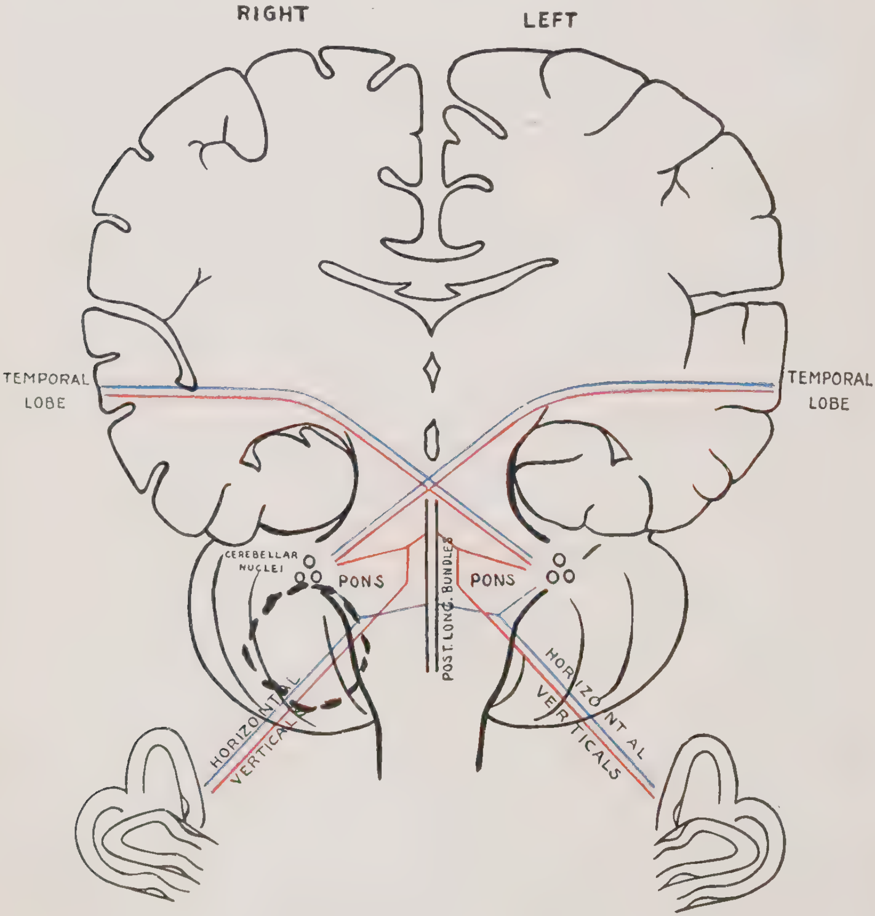


Fig. 128.—Tumor in right cerebello pontile angle.

The ear-examination, however, was useful not merely in corroborating the neurologic diagnosis, but also in furnishing additional information as to the extent of the lesion. It will be noted that under no conditions were we able to produce a past-pointing of the right arm to the left no matter what type of stimulation was employed. This suggested an actual involvement of the right cerebellar hemisphere. Stimulation of the left vertical semicircular canals produced practically no responses. This of itself would suggest an involvement of the left side of the pons. This involvement, however, was one produced by pressure rather than infiltration, because turning the patient both to right and to left with the head forward (in which position the vertical semicircular canals were stimulated) produced a good rotary nystagmus with falling, which distinctly demonstrated that the vertical canals pathways were not destroyed. Douching produced no responses because the caloric type of stimulation is much weaker. The autopsy corroborated the findings of the ear-examination.

CASE 30.—John B. K., age 39. Patient had been in perfect health until the spring of 1914, when he began to have occasional headaches. In September, 1914, as he was stepping off of a train, he was suddenly seized with violent vertigo, nausea and vomiting. The vertigo was so severe that he fell to the platform and was taken to the hospital, where he was confined to bed for ten days, during which time the vertigo gradually diminished. At the time of this attack of vertigo there was coincident complete deafness in the right ear. For the next two months his symptoms gradually improved and he was disturbed only by occasional staggering.

A careful neurologic examination in December, 1914, that is, three months later, failed to reveal any cause for staggering.

Ear examination, made at this time, showed complete destruction of the right cochlea and no responses whatsoever from any of the semicircular canals of the right ear. The Wassermann test was made at two different laboratories and from each laboratory came the report of a 4+ Wassermann. It was obviously from the ear tests that either the right labyrinth or VIII Nerve was destroyed. The diagnosis suggested, therefore, was a specific neuritis or labyrinthitis of the right side.

This diagnosis, however, was absolutely incorrect. There was noted, even on this first examination, a spontaneous vertical nystagmus upwards, and, in fact, moving pictures were taken of this nystagmus at the time.

Subsequent history of the case showed how badly we erred in not realizing fully the significance of spontaneous vertical nystagmus. As time went on his vertigo increased and the headache became worse. In December, that is, three months later, he noticed that his vision was failing and his loss of vision was becoming progressively worse. In April of the following year he began to cough. There was no pain in his chest and no expectoration, but this has gradually developed into hoarseness. He was losing weight and getting generally weak, so that in April of 1915 he applied for admission to the University Hospital.

The following notes of the physical examination at the hospital were made: "There is a marked impairment of the right apex with prolonged and harsh breath sounds. Few fine inspiratory râles. Tactile fremitus increased here. The rest of the lung is normal. Heart and abdomen negative.

"Eyes, slight exophthalmus. Pupils equal, react to light and distance. There is no nystagmus on looking straight ahead, but there is a lateral nystagmus on looking to either side. This is more marked when he looks to the right. There is a vertical nystagmus on looking upward. Paresis of the right external rectus. Both eye-grounds show choked disc—4 diopters in the right and 5 diopters in the left. There is a marked retinal œdema with a great amount of exudate and with numbers of retinal hemorrhages. The choking has evidently been present for some months. A rough test of the visual field shows no decided limitation.

"Nose and throat. Pharynx slightly congested. There is slight difficulty in swallowing. Large perforation of cartilaginous nasal septum. Loss of sensation of the right half of the soft palate. Left vocal cord moves easily, the right cord is stationary.

"No disturbance of sensation in the face. Adiadokokinesis in both hands, but far more marked in the right. Slight weakness of grip in the right hand. Sense of position in both arms normal. Sensation present on both sides. In finger-to-nose test there is slight ataxia on both sides. Reflexes exaggerated on both sides. Both knee jerks exaggerated. Babinski present on left, doubtful on right. No ankle clonus. On standing patient falls to left. When walking staggers to both sides, but especially to the left."

Wassermann reaction negative. X-ray examination negative.

Sputum examination negative.

Blood count practically normal except that there are 15,000 leucocytes.

Patient was operated on by Dr. Frazier, April 22, 1915. Bilateral cerebellar decompression. After the dura was opened, the right hemisphere bulged perceptibly, much more than the left. At this stage of the operation it was thought wisest to discontinue further exploration, until the structures could accommodate themselves to the relief of tension.

The patient later died and at autopsy a large cyst was found which apparently originated in the right cerebello-pontile angle and invaded the right cerebellar hemisphere. There was marked pressure against the brain-stem, which was displaced to the left.

## Comment

The diagnosis of specific labyrinthitis or specific involvement of the right VIII Nerve, made on the original ear-examination, was not only unfortunate but entirely unnecessary. The strongly positive Wassermann reaction apparently gave a clue to the original source of his trouble; subsequent Wassermann tests were entirely negative. As we look back on this case, however, we realize that it was not so much the lack of information at the time of the original examination, but our wrong interpretation of the findings which led to this incorrect diagnosis. Although the history of the onset of his original attack pointed to the diagnosis of a sudden destruction of the right labyrinth—as he gave the typical classical picture of the so-called "Meniere's disease," yet there were certain facts which could not have been explained on the basis of such a diagnosis:

(1) The spontaneous vertical nystagmus both upward and downward. Since the time of this examination we have come to learn that such a nystagmus cannot be caused by an involvement of either the labyrinth or VIII Nerve and that it is pathognomonic of an intracranial lesion and indicates an involvement of the brainstem either by infiltration or pressure.

(2) The patient had noticed a distinct diplopia for a full day following the original attack.

These two findings should have put us upon the right track and made us realize that we were not dealing with an end-organ lesion.

Case 31.—Mr. W. H., age 48. Patient was admitted to the University Hospital on January 26, 1916, with the chief complaint of headache, vertigo and staggering. He gave the following history: Three months before admission to the hospital he bumped his forehead. This was followed by dizziness, which rendered him unable to work. Headache came on a week later. These are constant, are chiefly frontal, but also extend to the top of the head and occiput. Although he had been hard of hearing for 15 years in the right ear, this has become a great deal worse since the onset of the trouble. Two months before admission he began to stagger, mainly to the left, but now he thinks that he staggers to the right instead. He noticed at this time that his vision was becoming poorer. He has some difficulty in swallowing liquids and he thinks that his voice is not what it ought to be.

The previous medical history, social and family history are negative.

He denies venereal history.

The chest, heart and lungs are normal.

The sense of smell is unimpaired. Sensation to pain and touch is normal over the face, trunk and extremities. The reflexes are slightly increased in both upper and lower extremities.

A lumbar puncture revealed a normal cerebro-spinal fluid.

The Wassermann is negative.

## CHART XLIV A

Name Mr. W. H. Age 48 Date Feb. 1, 1915.

Address

Referred by University Hospital

**DIAGNOSIS:**

**SUMMARY:**

**Complains of** Vertigo and staggering.

HISTORY:

Dizziness Yes
Staggering Yes
Deafness Yes
Tinnitus Yes

NOSE: Marked deviation of septum to the Right.

THROAT: Tonsils small Pharynx injected, paretic and anesthetic

EARS:
A. D. Negative
A. S. Negative

**Fistula** Negative

Hearing Tests

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 0 | A | 0 | Ac | | Bc | ? | n | Pol. | Negative | c⁴ | 0 | Galt | 0 | Weber |
| = | 2 | | 8 | Ac | > | Bc | = | n | | Negative | | Good | | 1.5 | Left. |

## CHART XLIV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

NYSTAGMUS

Looking to RIGHT ←
quick and marked
Looking to LEFT →
Slow and not as marked
Looking UP ↑
Looking DOWN ↓
Right cornea anesthetic

POINTING

| | RIGHT | LEFT |
|---|---|---|
| Shoulder from above | Touched | Touched |

Nystagmus present in all directions
Vertigo Yes
Past-pointing None
Falling Yes To Right
Romberg Considerable swaying
Turning head to right does not affect the swaying
Turning head to left " " " " "
Attempt to overthrow pelvic girdle does not react at all
Falls over like a wooden man.

---

### TURNING

To RIGHT →
Amp. Fair
Duration 15 Sec.

| | RIGHT | LEFT |
|---|---|---|
| To RIGHT<br>Shoulder from above | 3" to Left | 3" to Right. |

Nystagmus Impaired
Vertigo Impaired
Past-pointing Absent outward pointing of Right arm.

To LEFT ←
Amp. Fair
Duration 20 Sec.

| | RIGHT | LEFT |
|---|---|---|
| To LEFT<br>Shoulder from above | 5" to Left | Touched |

Nystagmus Impaired
Vertigo Impaired
Past-pointing Absent outward pointing of Left arm

---

### CALORIC

Douche RIGHT None
Amp. None
After 3 min. 20 sec.

| | RIGHT | LEFT |
|---|---|---|
| Douche RIGHT<br>Shoulder from above | Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back None
Amp.

| | RIGHT | LEFT |
|---|---|---|
| no vertigo | Touched | Touched |

Douche LEFT ↶
Amp. Fair
After 1 min. 10 sec.

| | RIGHT | LEFT |
|---|---|---|
| Douche LEFT<br>Shoulder from above | 2" to Left | 2" to Left |

Nystagmus Normal
Vertigo Subnormal
Past-pointing Normal
Falling Normal

Head Back ←
Amp. Good

| | RIGHT | LEFT |
|---|---|---|
| | 4" to Left | 4" to Left. |

*Eye Examination by Dr. Shumway.*—Pupils equal and respond promptly. Ocular movements restricted. Eye grounds entirely healthy.

Dr. Charles K. Mills dictated the following notes: "There is a marked Romberg both with the eyes open and shut. He can scarcely stand without support. Staggers to the right in an awkward effort to walk. There is present a marked nystagmus in every direction in which he attempts to look. The right palpebral fissure is wider than the left. The horizontal wrinkles of his forehead are somewhat more extended to the left side and he does not draw up the right angle of the mouth as strongly as the left. In attempting to whistle or to blow with his mouth, the oral movements are more positive on the left side. Patient has a very distinct dysarthria. The speech is what might be termed a bulbar, paretic kind. He also has direct difficulty in swallowing. When he attempts to drink water it goes down with difficulty and regurgitates slightly. The soft palate moves scarcely at all. With it there is an anesthesia of the soft palate and pharynx, which is bilateral just the same as the palsy. The voice is distinctly nasal. The patient shows distinct adiadokokinesis, particularly in the right upper extremity. There is dysmetria in the finger-to-nose test which is very marked in the right and shows itself as an undershooting (hypometria)."

An examination of the vestibular apparatus was made on February 1, 1915, and the data obtained shown on chart XLIV.

It will be noted that the hearing is good on the left side and totally gone on the right.

There is a spontaneous nystagmus in every direction but more marked upward and downward. The pelvic girdle reactions are very poor—the patient falls over like a broomstick at the slightest touch.

Turning produces an impaired nystagmus and what may be termed "crossed" past-pointing. Turning to the right and turning to the left always produces a past-pointing of both arms inward—in other words, there is always an absence of the outward past-pointing of both arms after turning.

The caloric test shows a dead, non-responsive ear on the right side and normal responses on the left.

The presence of a vertical nystagmus, a stiff pelvic girdle and the peculiar past-pointing after turning are of themselves sufficient to indicate a central lesion without any reference to the neurologic data in this case. The absence of all responses from the right ear indicate a peripheral lesion. This combination of a central and peripheral lesion can obviously be best explained by a lesion situated in the cerebello-pontile angle.

The impaired nystagmus obtained on stimulation would suggest an involvement of the brain-stem itself and the definite impairment of past-pointing, together with the poor pelvic girdle reactions, would similarly suggest an involvement of the cerebellum.

The patient was operated on on March 2, 1915, and unfortunately contracted an inspiratory pneumonia, of which he died a week later.

The following is the autopsy report:

*Head.*—Upon removing the brain case the meninges are taut, blood vessels congested. Cutting through the dura there is some increase in the amount of fluid.

On lifting the frontal lobe to the right side two nodular swellings are noticed; one lies within the petrous portion of the temporal bone in front of the ridge of the sella turcica. This nodule is about 1.5 c.m. in diameter, is rounded and upon its surface is covered by the dura, which is perfectly smooth over the entire mass; apparently the tumor is growing between the dura and the bony portion of the petrous. The second nodule is larger, more rounded, bulges more, is cystic to the touch and lies posterior to the petrous portion and beneath the tentorium cerebelli. Upon incising the tentorium in an area between the two tumors the knife apparently went through a peduncle with the escape of fluid, at least an ounce; this fluid apparently coming from beneath the tumors, much more from the larger one, in

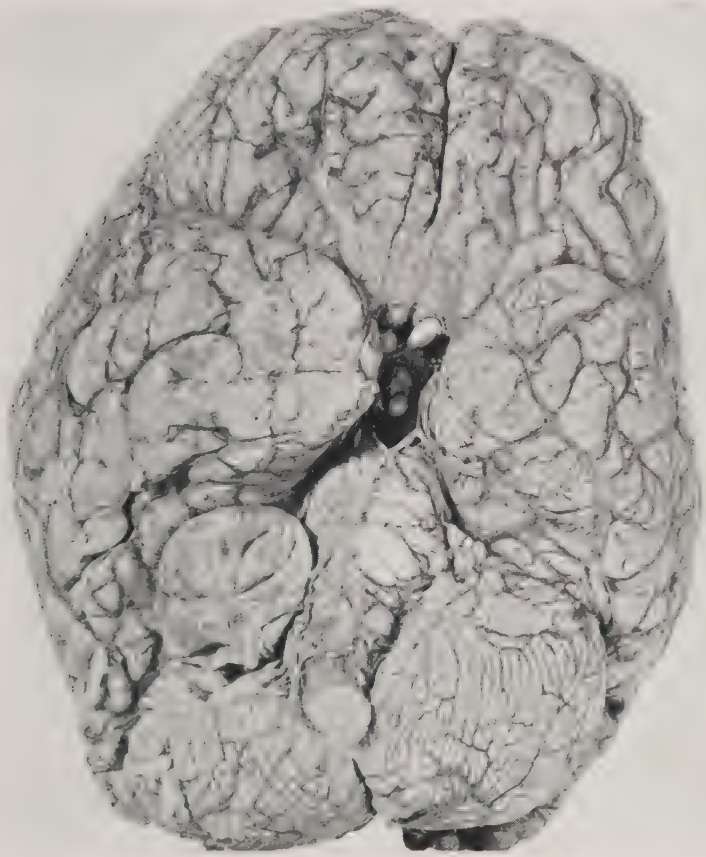


FIG. 129.—Large cyst in right cerebello-pontile angle.

which it was apparently under pressure. The second tumor, that is, the one beneath the tentorium, was removed intact with the brain. The first one in the petrous portion was dissected out. This apparently occupies a position which corresponds to the Gasserian ganglion upon this side. It is not, however, as near as can be made out by dissection, in direct communication with any nerve trunk. As described, it is completely covered by dura upon the upper surface, which is smooth; its under surface is in intimate connection with the bony portion of the temporal bone, from which it is necessary to free it by cutting, leaving a ragged edge both on the tumor and on the bony portion. The tumor was preserved in Orth's fluid, half of it used for section and the other half turned over to Dr. Spiller.

*Brain.*—The lateral ventricles opened and choroid plexuses removed. One section made through the cyst wall. This term has not been used before, but it refers to the second tumor described. In the fresh stage it is seen that this tumor is apparently a protrusion of the dura by fluid in this position into the cerebello-pontine angle. Here it presses upon the cerebellum, cerebrum and upon the pons and medulla oblongata. While there are a few small adhesions, it is apparently not in intimate relation with any of these structures. Before fixing, the cyst was filled, the opening tied off and in its present condition it is about half its original size.

*Notes dictated by Dr. Spiller:*

"The right VII and VIII Nerves are both implicated in the wall of the cyst; the acoustic more so than the facial. The former enters into the substance of the cyst wall, while the latter lies superficially just beneath the surface of the cyst wall. The glossopharyngeal and vagus nerves escape. Judging from the external appearance of this cyst it is merely a part of a cystic degenerated tumor. The fluid has now entirely escaped and palpation reveals a mass within the cyst that is apparently tumor. This is of nodular formation. There is no evidence of the V Nerve making an exit from the pons. At the part where the nerve should leave the pons are strands of fibres spread out over the surface of the mass and having the appearance of nerve fibres. These probably are fibres of the V Nerve. The cystic tumor lies directly in the cerebello-pontine angle. This has led to atrophy of the anterior and lower part of the right cerebellar lobe and to atrophy of the right half of the pons." (Fig. 129.)

CASE 32.—Mr. William L. S., age 33. Was admitted to the University Hospital on October 11, 1915, complaining of blindness and headache.

Three years ago he was struck on the head by a brick which fell from a scaffold three stories high. He was unconscious for about ten minutes, but then went to work and noticed no other bad results of the blow. Two years later he began to have attacks which he describes as "drawing" of the left side of the face. They begin at the sterno-clavicular articulation and end far up over face and forehead. These attacks appeared once or twice a day, lasted only a few minutes and were accompanied by twitching of the right upper and lower eyelids. Once or twice a week he would "fall." This falling was not to either side, but he just "crumpled up." About the same time the eyesight began to fail, and three weeks ago he became totally blind. Hearing has been poor in the left ear for four years. He has not had a drawing spell for over a month. His appetite is good, the bowels are regular. He sleeps poorly, that is, only a few hours at a time.

Questioning him on another day, the patient contradicted his original statement that the attack began with a drawing at the left sterno-clavicular articulation. Instead, he says that these drawing attacks began in his left cheek.

Family history negative.

Patient has been married for fifteen years and has six children, all of whom are well. Two children died in infancy.

*Physical Examination.*—Patient is large and well developed. Gait and station fairly good. No Romberg. No distinct weakness of facial muscles. Tongue is protruded in midline. The resistance of the patient to the opening of his lids is distinctly less on the left eye than on the right, and he does not draw up the left corner of his mouth as well as the right. He wrinkles his left forehead less than the right side of the forehead.

*Eye Examination by Dr. de Schweinitz.*—Extra-ocular muscles normal. Pupils react to light and convergence. Right eye: Light perception only, chiefly on the nasal side. There is apparently a subsiding choked disc with atrophy in its deeper layers. The arteries are small and the surface of the disc is raised three diopters. Left Eye: There is light perception, correctly projected on all sides. There is also choking of the disc, which is apparently subsiding. There are distinct signs of atrophy. Surface of the disc is raised three diopters.

Heart and lungs negative. Mouth and throat negative.

Upper extremities are well developed. No impairment of sensation to pain and touch. The reflexes, especially the biceps, are diminished on both side. Coordination is good with the right arm, but not so good with the left. There is a distinct tendency to hypometria with the left hand.

Lower extremities. Power of muscles good. Sensation to pain and touch normal. Patellar reflexes equally exaggerated. Achilles absent. No ankle clonus. No Babinski. Many scars on the ankles and feet. Several small chronic ulcers.

Wassermann negative.

An X-ray of the skull shows that there is possibly some depression to the right of the midline in the vault, but this is not definite. No evidence shown of tumor anywhere.

Repeated tests for the corneal reflex showed it to be absent on the left side and normal on the right.

The neurologic diagnosis in this case was a tumor of the right cerebral hemisphere implicating the face centre.

An examination of the vestibular apparatus was made on October 20, 1915, and the findings presented on the accompanying chart (XLV).

It will be noted that the hearing in the right ear is normal and that the left ear is stone deaf.

There was a spontaneous vertical nystagmus upward and a spontaneous past-pointing of both arms to the left.

Turning to the right produced a poorer and less prolonged nystagmus than turning to the left, which indicated that the right ear functionated more than the left. Turning produced a past-pointing of each arm in both directions, which suggested that the cerebellar cortex was probably normal.

## CHART XLV A

Name Wm. L. S. Age 33 Date Oct. 20, 1915.
Address
Referred by

DIAGNOSIS:

SUMMARY:

Complains of Blindness and headache

HISTORY:
Dizziness Occasional
Staggering Slight
Deafness in Left ear, 5 years.
Tinnitus

NOSE: Not examined

THROAT: Not examined

EARS: A. D. Drum head normal
A. S. Drum head normal

Fistula

Hearing Tests

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| A | 2 | A | 12 | Ac | > | Bc | = | n | Pol. | | $c^4$ | Good | Galt | 1.2 |
| = | 0 | | 0 | Ac | ? | Bc | | n | | | | 0 | | |

Left ear completely deaf.

## CHART XLV B

### TESTS OF THE VESTIBULAR APPARATUS

### SPONTANEOUS

**NYSTAGMUS**

Looking to RIGHT None
Looking to LEFT →
Looking UP ↑
Looking DOWN None

Slight paresis Right external rectus

Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 1" to Left | 1" to Left |

Nystagmus upward; to Left
Vertigo None
Past-pointing both arms to Left
Falling None
Romberg Slight swaying
Turning head to right Increases swaying.
Turning head to left " "
Attempt to overthrow Pelvic girdle reactions fair

---

### TURNING

To RIGHT →
Amp. Small
Duration 14 Sec.

To RIGHT
Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 4" to Right | 3" to Right. Touched |

Nystagmus Reduced
Vertigo present, short duration.
Past-pointing

To LEFT ←
Amp. Good
Duration 17 Sec.

To LEFT
Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 12" to Left | 8" to Left |

Nystagmus Normal
Vertigo Normal
Past-pointing Normal

---

### CALORIC

Douche RIGHT →
Amp. barely a twitch
After 2 min. sec.

Douche RIGHT
Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 3" to Right<br>Touched | 3" to Right.<br>Touched |

Nystagmus perverted
Vertigo subnormal
Past-pointing present
Falling subnormal

Head Back →
Amp. Large

| POINTING RIGHT | POINTING LEFT |
|---|---|
| 5" to Right. | 6" to Right. |

Douche LEFT None
Amp.
After 3 min. sec.

Douche LEFT
Shoulder from above

| POINTING RIGHT | POINTING LEFT |
|---|---|
| Touched | Touched |

Nystagmus None
Vertigo None
Past-pointing None
Falling None

Head Back None
Amp.

| POINTING RIGHT | POINTING LEFT |
|---|---|
| Touched | Touched |

Douching the right ear produced no nystagmus, but did produce past-pointing. On another examination several days later a nystagmus was produced, but this nystagmus was horizontal instead of rotary. This variability in the responses as well as the presence of the perverted nystagmus indicated pressure against the brain-stem. Putting the head back after douching produced a large horizontal nystagmus to the left, with normal past-pointing of both arms.

Douching the left ear with head upright and head back produced no responses whatsoever. This suggested a complete destruction of either the labyrinth or the VIII Nerve on that side.

The following report of the ear examination was made:

Left Ear.—(1) Cochlear fibres destroyed. No hearing whatever. (2) All kinetic-static fibres destroyed. Douching all canals gave no nystagmus, vertigo or past-pointing. The left VIII Nerve or labyrinth destroyed.

Right Ear.—(1) Cochlear fibres normal. (2) Horizontal canal pathways normal. (3) Vertical canals; no reaction.

*Conclusion.*—Right labyrinth, right VIII Nerve normal. Right side of medulla oblongata normal. Right side of pons involved.

*Summary.*—The best explanation of the above findings would be a neoplasm of the left cerebello-pontile angle pressing against the pons sufficiently to involve the vertical canals fibres from the opposite side.

Because this report conflicted with the neurologic diagnosis, the patient was again examined by the neurologic staff and the following notes were dictated:

Testing carefully for ataxia in the finger-to-nose test fails to show any reliable evidence. Adiadokokinesis tested by the revolution of the hand at the wrist or placing first the palm and then the dorsum of the hand on the thigh is entirely normal in the left as well as the right hand. Synergic movements in both upper limbs are entirely normal; also synergic movements in each lower limb. Cerebellar catatonia may be present. Is able to preserve the position for 4 minutes with slight swaying toward the end of the time. In bending backward at the waist he flexes his knees proportionate to the bending, in the normal manner. The left patellar tendon reflex is a trifle prompter than the right. The left Achilles tendon reflex is a trifle prompter than the right. Whereas the ear findings suggest a left cerebello-pontile lesion, other neurologic findings than those of the Barany tests do not confirm this localization. The Jacksonian convulsion with involvement later of the right eyelid indicates a right motor cortical irritation. The left facial paresis because of its involvement of the upper branch of the nerve could be cortical in origin, but, as it is probably not of recent development, it is more likely a peripheral paralysis. The examination suggests two lesions, an old one in the left cerebello-pontile angle likely to be circumscribed tuberculous meningitis and a more recent lesion in the right frontal motor cortex, more irritative than paralyzing. It may be that there are adhesions as to the result of the severe blow and as the result of increased intracranial pressure diminishing the resistance of the brain. This area of cicatrization is capable of acting as an irritant focus. The corneal reflex on the left side is unquestionably diminished and slightly diminished on the right. It must be remembered, however, that there is some weakness of the left orbicularis palpebrarum so that a stronger afferent impulse from the conjunctiva will be necessary to cause contraction of the paretic muscles. The response to the left orbicularis palpebrarum to slight touch of the left eyelash is exceedingly prompt.

As originally planned, an operation decompressing the right frontal lobe was done on November 29, 1915, and no lesion discovered.

Patient died three months later and autopsy revealed a large tumor in the left cerebello-pontile angle.

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